Ano 1919

Tomo XI

Faciculo II

Translations

MEMORIAS
DO
INSTITUTO OSWALDO CRUZ

Rio de Janeiro - Manguinhos

Digitized by the Internet Archive
in 2025

https://archive.org/details/memorias-do-instituto-oswaldo-cruz_1919_11_2

Ciliés Intestinaux de Mammifères

par le

DR. ARISTIDES MARQUES DA CUNHA,

Assistant à l'Institut Oswaldo Cruz.

(Avec Planche 1.)

Il y a longtemps que nous étudions les ciliés qui se trouvent dans le tube digestif des Mammifères et qui y habitent en commensaux. Les Mammifères, hôtes de ces protozoaires, sont en général des herbivores; les ciliés se trouvent dans l'estomac chez les Ruminants et dans le caecum chez les Non-Ruminants.

Leur présence n'a pas été constatée chez tous les Herbivores, car ils ne se trouvent que là, où l'alimentation de leur hôte leur convient et généralement chez les animaux qui se nourrissent de graminées.

Nous savons que les affinités zoologiques entre les ciliés parasites des différentes espèces de Mammifères dépendent plutôt de la manière de se nourrir de leurs hôtes que de leur position systématique; cela est démontré par l'exemple du cheval et de la capyvara *(Hydrochoerus capibara)*, qui tous les deux sont hôtes des ciliés des genres *Cycloposthium* et *Paraisotricha* quoique l'un soit Ongulé et l'autre Rongeur. On ne s'étonne donc pas de trouver que le tapir ou anta (*Tapirus americanus)*, vu son genre de vie, soit l'hôte de ciliés de position systématique voisine à celle des ciliés qui se trouvent chez les susdits animaux.

Le Dr. OLYMPIO DA FONSECA, membre d'une mission scientifique, voyageant dans le Brésil et dans les pays voisins pour faire des recherches, eût l'occasion d'examiner des matières provenant du tube digestif d'un tapir et y trouva des ciliés de l'espèce dont il s'agit. Il nous envoya les matières bien conservées; nous profitons de cette occasion, pour lui témoigner notre reconnaissance, ainsi qu'à ses collègues.

Les matières où se trouvaient les ciliés, provenaient du caecum du tapir et furent fixées en alcool sublimé de SCHAUDINN; nous en fimes des préparations colorées au borax-carmin et à l'hématoxyline de DELAFIELD qui servivent à l'étude des protozoaires observés.

Le matériel était très-riche en ciliés et, á côté de quelques individus qui ne pouvaient être identifiés par les détails de structure, observés dans les préparations colorées, il y avait une espèce plus abondante et mieux

conservée qui se prêtait bien à l'étude et qui forme l'objet du présent travail.

Nous avons déjà signalé cette espèce dans une note préliminaire en créant pour elle le nouveau genre *Prototapirella*, inclu dans la famille *Cycloposthiidae.*

Voici la diagnose et une description détaillée de l'unique espèce de ce genre actuellement connue:

Prototapirella CUNHA, 1918.

Diagnose: "Cycloposthiide qui en sus des deux stylets caudaux postérieurs trouvés sur les côtés du corps, en a deux autres sur le bord dorsal." La collocation dans cette famille est justifiée par la grande ressemblance de notre espèce avec le genre *Cycloposthium* dont elle se distingue à peine par le nombre des stylets caudaux.

Espèce type: *Prototapirella intestinalis* CUNHA, 1915.

Corps cylindrique, très-aplati latéralement, avec un bord ventral droit ou légérement concave et bord dorsal convexe, montrant deux dépressions où s'insèrent les deux stylets caudaux du dos. L'extrémité antérieure est tronquée; la postérieure présente deux dépressions (une de chaque côté) où s'insèrent les deux stylets caudaux postérieurs; la partie qui fait suite à la dépression est plus étroite, arrondie et généralement courbée vers le bord ventral.

A l'extrémité antérieure se trouve le péristome, dont la formation est tout-à-fait analogue à celle du *Cycloposthium.* Au dehors, il consiste en une saillie incolore qui est la prolongation des parois latéraux du corps; après la saillie se trouve un sillon plus ou moins profond, selon l'état de rétraction du péristome.

Plus en dedans, il y a une autre saillie, le ciliophore qui a, á peu près, la forme d'un cône tronqué; à son extrémité s'ouvre la bouche, suivie du cytopharynx très-court; autour de la base du ciliophore s'insère la couronne de membranelles.

Le péristome est rétractile et le ciliophore s'invagine entre les bords circulaires dont nous avons déjà parlé; ceux-ci prennent la place, occupée auparavant par le ciliophore s'inclinant en dedans. La contraction du ciliophore se produit au moyen de myofibrilles, qui s'insèret à la base de celui-ci et se dirigent en arrière (Voir Pl. I, fig. 3).

En outre des membranelles du péristome il y a aussi des touffes de membranelles autour des stylets caudaux, qui forment l'appareil locomoteur.

Il y a quatre stylets caudaux, deux postérieurs comme ceux du *Cycloposthium* et deux dorsaux, situés sur le bord dorsal du cilié. Les stylets caudaux postérieurs s'insèrent dans les deux dépressions près de l'extrémité postérieure, un sur chaque bord du corps, de manière à ce que celui du bord ventral se trouve toujours un peu en avant de celui du bord dorsal, ce qui est dû a la susdite courbature de l'extrémité vers le bord ventral. Nous donnons à ces stylets le nom de postérieur ventral, ou tout simplement ventral et dorsal postérieur.

Les deux autres stylets se trouvent au bord dorsal dans les dépressions dont nous avons parlé: l'un dorsal antérieur dans le tiers antérieur, près du péristome et l'autre dorsal médian dans le tiers du milieu, en arrière du centre du corps et même quelque fois au point de contact entre les tiers médian et postérieur. Les stylets ont, comme ceux du *Cycloposthium*, une partie basale en forme d'une petite saillie cylindrique, où s'insère une touffe de membranelles. Il est inutile de décrire la structure intérieure qui ne se distingue pas de celle du *Cycloposthium.*

Le macronucléus est allongé en forme d'écharpe. Sa partie antérieure plus large se rétrécit brusquement au niveau de la dépression médiane, mais elle s'étend jusque près de l'extrémité caudale où elle peut être un peu dilatée. Elle se trouve près du bord dorsal et s'étend entre le stylet caudal antérieur et le postérieur du dos, dépassant ce dernier. Vu de côté, le macronucléus accompagne le bord dorsal, sa partie postérieure étant légèrement déviée vers la face ventrale. Vu d'en haut, il montre une saillie au milieu de la partie antérieure plus épaisse qui s'étend latéralement

et occupe la partie convexe entre les dépressions antérieure et moyenne du bord dorsal.

Au milieu de la partie épaissie du macronucléus et au point même ou il se bifurque, il y a une dépression où se loge le micronucléus. Sa forme est généralement allongée, ellipsoide ou fusiforme, rarement sphérique comme un des micronucléi de la phase de division, reproduite en fig. 3 Pl. 1. La forme ronde est plus commune dans les micronucléi provenant d'une division récente

Les vacuoles contractiles se trouvent auprès du borp dorsal, mais nous n'en avons pu déterminer exactement le nombre, vu que nous n'avons pas eu l'occasion e'examiner ce cilié en état frais.

Les dimensions du cilié sont assez variables; la longueur oscille entre 80 et 140 μ, quelques individus atteignent 180 μ; leur largeur varie entre 60 et 80 μ et peut s'élever jusqu a 120 μ. La longueur du grand noyau varie de 60 a 80 μ, la plus forte largeur de la partie la plus grosse est près de 10 μ.

Dans le matèriel étudié, on voit quelques formes de division du cilié (Fig. 3). On y peut constater la présence de deux micronucléi, provenant de la division du micronucléus primitif, ainsi que le sillon de séparation des corps. L'espace clair près du bord ventral, derrière les sillons, présente le péristome en voie de formation dans un des ciliés provenant de la division.

Je profite de cette occasion, pour dire quelques mots sur la position systématique des ciliés parasites des Mammifères, car nous ne sommes point d'accord avec les auteurs qui se sont occupés de cette question. Par exemple, HICKSON et POCHE incluent *Didesmis* dans la famile *Cycloposthi:dae*, malgré que ce genre n'aît pas le péristome rétractile de *Cycloposthiium* et que ce genre aît des cils longues et fins au lieu de membranelles; leur structure intérieure est absolumment différente. Il nous paraît beaucoup plus raisonnable de porter l'espèce *Didesmis* à la famille *Buetschliidae* parcequ'elle montre bien d'analogie avec les représentants de cette famille.

Il en est de même avec le genre *Blepharocorys* qui ne doit pas appartenir à la famille *Colpotidae*, pas plus que *Paraisotricha* à celle des *Isotrichidae*. Nous sommes d'accord avec BUNDLE, qui leur établit une famille à part. A cette famille, on pourrait donner le nom de *Paraisotrichidae*, comme nous l'avons fait, dans une autre publication.

Suit une liste des Ciliés parasites des Mammifères avec leur position systématique par rapport aux modifications exposées plus haut et en y inscrivant les espèces et les familles tout dernièrement décrites.

Ordre Holotricha.

Sous-ordre Stomata.

Famille: Buetschliidae POCHE 1913.

Buetschlia SCHUBERG, 1888.
Didesmis FIORENTINI, 1890.
Blepharoposthium BUNDLE, 1895.
Blepharocodon BUNDLE, 1895.
Blepharosphaera, BUNDLE, 1895.

Famille: Istrochidae SCHOUTEDEN, 1906.

Isotricha STEIN, 1858.
Dasytricha SCHUBERG, 1888.

Famille: Paraisotrichidae CUNHA, 1916.

Paraisotricha FIORENTINI, 1890.
Blepharocorys BUNDLE, 1895.

Famillie: Cyathodiniidae CUNHA, 1914.

Cyathodinium CUNHA, 1914.

Sous-Ordre: Astomata.

Famille: Enterophryidae HASSELMAN, 1918.

Enterophyra HASSELMAN, 1918.

Ordre Pycnotrichidea POCHE, 1913.

Famillie: Pycnotrichidae POCHE, 1913.

Pycnothrix SCHUBLATZ, 1908.

Ordre Heterotricha.

Famille: Plagiomatidae POCHE, 1913.

Nyctotherus LEIDY.

Famille: Bursariidae KENT, 1880.

Bolantidium CLAPAREDE & LAMAN, 1858.

Ordre Oligotricha.

Famille: Orphryscolecidae CLAUS, 1874.

Orphryoscolex STEIN, 1858.

Entodinium STEIN, 1858.

Diplodinium SCHUBERG, 1888.

Troglodítella BRUMPT & JOYEUX.

Metadinlum AWERINZEW & MUTAFOWA.

Cunhaia HASSELMAN, 1918.

Famille: Cycloposthiidae POCHE. 1913.

Cycloposthium BUNDLE, 896.

Prototapirella CUNHA, 1911.

Explication de la Planche 1.

Toutes les figures furent dessinées de préparations colorées au carmin boracique, avec usage de la chambre claire à la hauteur de la table et avec agrandissement de diamètre.

Fig. 1. Prototapirella intestinalis CUNHA

Fig. 2. Macronucléus de la *Prototapirella intestinalis* vu en sens dorso-ventral, démontrant la prolongation latérale de la même.

Fig. 3. Forme de division du cilié.

On the Brazilian Species of the Sub-family Subulurinae TRAVASSOS, 1914,

by

DR. ANTONIO LUIS DE B. BARRETO. (1)

(With Plates 2--24).

Material.

The material used for this study forms part of the helminthoiogical collection belonging to the «Instituto Oswaldo Cruz», nos. 1070 to 11 34. To the specimens already there existing, the greater part of which were collected by Drs. A. LUTZ and L. TRAVASSOS, I add those obtained from 500 autopsies made by me.

Sub-family SUBULURINAE Travassos, 1914.

Synonymy.

Subulurinae TRAVASSOS, 1914 – p. 137.
Kathlaniinae LANY, 1914 – p. 655.
Subulurinae HALL, 1917 – p. 51.

History.

In 1914, TRAVASSOS established the sub-family *Subulurinae*, in Nematology, thus elevating to the category of sub-family the nematodes until then included in the genera *Subulura* and *Oxynema* taking as type especies, *Subulura* MOLIN of which he gives the following diagnosis:

Mouth with three indistinct lips, or without them, followed by a vestibulum. Oesophagus with bulb; males mith fusiform sucker without chitinous rim. Spicules unequal accompanied by a gubernaculum. Not knowing the work of TRAVASSOS, and noticing that the number of *Heterakidiae* with sucker without chitinous rim, is quite considerable, LANE established the sub-family *Kathlaniinae* which comprises the genera *Subulura*, *Dacnitis*, *Cissophyllus* and *Kathlania* n. gen., calling attention to the fact that there are always 11 pairs of papillae on the tail of the males in this new sub-family.

After having carefully studied some parasites of the genus *Subulura* and looked through the literature, we think we are jus-

(1) Handed in for publication in December, 1917. However, as new studies appeared by degrees, we completed this monograph with the literature on the subject until the year 1916.

tified in differing from LANE, as we can show that 11 pairs of genital papillae are not always present in the species of the new sub-family proposed by this author.

Even in the genus *Subulura* such unswerving unity has not been confirmed, the variations in number and position of the caudal papillae serving to individualise the species of this genus.

In an interesting monograph on the worms of rodents, and while speaking of *Subulurinae* HALL asserts that he prefers the determination of TRAVASSOS to that of LANE, especially on account of its type being an older and more known genus.

Agreeing with HALL, we confirm his decision, and are of opinion that TRAVASSOS' name must be considered as final on account of its priority, as his 3rd. "contribution to the knowledge of the Helminthological Fauna of Brasil" dates from February 1914 while LANE's publication only appeared in October of the same year.

Diagnosis: *Acaroidea, Heterakidae Polymiariae.* Mouth elliptic or polygonal with two or three indistinct lips. Oesophagus always followed by a bulb. Males with spindle-shaped or oval sucker without chitinous ring; spicules rarely absent *(Heteroxynema)*; generally one or two of them, equal or unequal, always accompanied by an accessory picce.

Type genus: *Subulura* MOLIN, 1860.

Systematic Position.

At present, the super-family Ascaroidea thus established, comprises:

- *Ascaroidea* Railliet & Henry, 1915
  - *Ascaridæ* Cobbold, 1864.
  - *Heterakidæ* Railliet & Henry, 1913
    - *Heterakinæ* Railliet & Henri, 1912
    - *Subulurinæ* Travassos, 1914
    - *Seuratinæ* Hall, 1916
  - *Anisakidæ* Railliet & Henry, 1915
  - *Heterocheilidæ* Railliet & Henry, 1915.
  - *Cucullanidæ* Barreto, 1916.

Genera.

According to TRAVASSOS, the sub-family *Subulurinae* comprises 2 genera: *Subulura* MOLIN 1899 and *Oxynema* LINSTOW 1899.

LANE is of opinion that it comprises the following genera: *Kathlania* n. gen. (type genus) *Subulura* MOLIN 1860, *Dacnitis* DUJ. 1845 and *Cissophyllus* RAILLIET & HENRY 1912.

HALL thinks that the genus *Kathlania* LANE, 1914 ought to be separated from the *Subulurinae* perhaps to range as a new subfamily; he considers *Subulura* MOLIN, 1860 as type genus, and includes in this sub-family *Heteroxynema* n. gen.

We now include in the sub-family *Subulurinae* the five following genera: *Subulura* MOLIN, 1860 *Oxynema* LINSTOW, 1899 *Cissophyllus* RAILLIET & HENRY, 1912, *Heteroxynema* HALL 1916, and *Numidica* n, gen. Of these some are only classified temporarily, as for instance: *Cissophylius* and *Heteroxynema.*

Unfortunately, we have not yet had any opportunity of examining material of *Chelonians* nor specimens of *Heteroxynema.* We cannot, therefore, form a definite opinion as to the exact position, to be occupied by these two genera in the classification of nematodes.

We are however inclined to think, judging from the descriptions and illustrations of the above-named authors, that the genus *Cissophillus* must be allied with the *Cucullanidae* RAILLIET & HENRY 1916; further, that the *Heteroxynema* of HALL, whose male specimens has absolutely no genital organs, can only be determined when fully developed males are described, for we are under the impression that this specimen was not an adult.

As to the genus *Kathlania* LANE, 1914 we classify it with the *Subulurinae* on account of the general aspect of the body and especially of the conformation of the cephalic end of the worms of this genus. In his description of the species, LANE omits to speak of the disposition of the muscular ele-

ments, and for this reason, we cannot say for sure, in what group this new genus should be placed.

Should the study of the contractile layer reveal muscles of the meromyarian type of SCHNEIDER, the transfer to the family of *Oxyuridae* is justified: if it be of the polymayrian type with muscular cells, it is better, to put the genus apart as a sub-family as LANE does.

In a former communication (BARRETO 1916) we excluded the genus *Cucullanus* MULLER 1777 (*Dacnitis* DUJ. 1845) from the *Subulurinae*, making it an independent family *Cucullanidae* BARRETO 1916 (*Dacnitis* LANE, 1914).

Table for the rapid diagnosis of the genera of the Sub-family SUBULURINAE.

I. Spicules absent. – *Hetroxynema.*
II. Spicules present.
1. One spicule. – *Oxynema.*
2. Two spicules.
A. Mouth with three lips, with chitinous edges *Cissophyllus.*
B. Mouth with indistinct lips, without chitnous edges.
a Sucker fusiform, elliptic without chitinous formation. – *Subulura.*
b Sucker oval, with small trabeculae of chitin. – *Numidica.*

Of the genus SUBULURA MOLIN, 1860.

Synonyms.

Ascaris pro parte LINNAEUS, 1756.
Heterakis pro parte DUJARDIN, 1845 p. 222.
Oxyuris pro parte CREPLIN, 1853 – p. 59.
Subulura MOLIN, 1860 – p. 352.
Allodapa DIESING, 1860 – p. 644.
Subulura DIESING, 1860 – p. 644.
Subulura COBBOLD, 1864 – p. 67.
Allodapa COBBOLD, 1864 – p. 67.
Heterakis pro parte SCHNEIDER, 1866 – p. 66.
Subulura DRASCHE, 1882 – p. 120
Heterakis acheilostomi pro parte STOSSICH, 1888 – p. 278.
Heterakis acheilostomi pro parte RAILLET, 1895 – p. 409.
Subulura RAILLIET & HENRY, 1912 – p. 258.
Subulura RAILLIET & HENRY, 1913 – p. 679.
Subulura TRAVASSOS, 1913 – 1913 (a,) p. 2 e 18.
Subulura TRAVASSOS, 1913—1913 (b,) p. 272 e 297.
Subulura TRAVASSOS, 1914—p. 138.
Subulura SEURAT, 1914—1914 a., p. 154.
Allodapa SEURAT, 1914—1914 (b.) p. 195.
Subulura pro parte HALL, 1916—p. 51 – 52.
Allodapa HALL, 1916—p. 52 and 60.

Synopsis of previous work on the subject.

The genus *Subulura* was established in 1860 by MOLIN for a nematode found in the intestine of *Scops brasilianus*, and which he called *Subulura acutissima.*

Several species, described before by the helminthologist RUDOLPHI, as belonging to the genus *Ascaris* LINNÉ and subsequently considered as *Heterakis* by DUJARDIN, ought now to be included in the genus *Subulura.*

In 1860, DIESING, although maintaining this genus as the species *acutissima* admitted a new genus (*Allodapa*) for the *Oxyuris allodapa* of CREPLIN, a parasite of the caecum of *Cariama cristata*, and closely related to *Subulura acutissima.*

In 1864 COBBOLD adopted DIESING'S way of thinking.

Later on, (1866) SCHNEIDER, without taking MOLIN and DIESING's genera into account, identified the species *acutissima* and

allodapa, with *Ascaris strongylina* (RUDOLPHI) including it in the genus *Heterakis* DUJ. and thus doing away with the genera *Subulura* and *Allodapa*.

In his description of *Subulura acutissima* DRASCHE (1882) suggests that this species must belong to DUJARDIN'S genus.

In 1888 STOSSICH shows the necessity of dividing the genus *Heterakis*, and proposes the separation into two sections: *Cheilostomi* and *Acheilostomi*: this division was for some time accepted (RAILLIET), the specimens of the genus *Subulura* being classified in the latter group.

LIST OF SPECIES OF THE GENUS SUBULURA

SPECIFIC NAME	AUTHOR	DATE	GEOGRAPHIC DISTRIBUTION
1. *Subulura distans*.	Rudolphi	1809	Africa.
2. *Subulura subulata*.	Rudolphi	1819	Europa (Spain).
3. *Subulura forcipata*.	Rudolphi	1819	America (Brazil).
4. *Subulura strongylina*.	Rudolphi	1819	America (Brazil).
5. *Subulura reclinata*.	Rudolphi	1819	America (Brazil).
6. *Subulura allodapa*.	Creplin	1853	America (Brazil).
7. *Subulura jacchi*.	Marcel	1857	America (Brazil).
8. *Subulura acutissima*	Molin	1860	America (Brazil).
9. *Subulura (?) annulata*.	Molin	1860	America (Brazil).
10. *Subulura suctoria*.	Molin	1860	America (Brazil).
11. *Subulura papillosa*.	Molin	1860	America (Brazil).
12. *Subulura (?) peramata*.	Ratzel	1868	
13. *Subulura (?) andersoni*.	Cobbold	1876	Asia (India).
14 *Subulura curvata*.	Linstow	1883	Asia (Turkestan).
15. *Subulura otolicni*.	v. Beneden	1890	Africa (Guinea).
16. *Subulura differens*	Sonsino	1890	Cosmopolitan species.
17. *Subulura sarasinorum*.	Meyer	1896	Asia (India, Ceylon).
18. *Subulura (?) gracilis*.	Linstow	1899	
19. *Subulura acuticauda*.	Linstow	1901	Africa (Usanga).
20. *Subulura recurvata*	Linstow	1901	Africa (Nyassa).
21. *Subulura rimula*.	Linstow	1903	Asia (Siam).
22. *Subulura rima*.	Linstow	1906	
23. *Subulura poculum*	Linstow	1909	Africa
24. *Subulura schebeni*.	Linstow	1909	Africa.
25. *Subulura leprincei*.	Gendre	1909	Africa (Guinea, Dahomey, Tunis).
26. *Subulura similis*.	Gendre	1909	Africa (Dahomey).
27. *Subulura elongata*.	Seurat	1914	Africa (Algeria).
28. *Subulura noctuæ*.	Seurat	1914	Africa (Algeria).
29. *Subulura (?) macronis*.	Stewart	1914	Asia (India).
30. *Subulura pigmentata*.	Gedoelst	1917	Asia (Sumatra).
31. *Subulura seurati*.	Barreto	1917	Africa (Algeria).
32. *Subulura halli*.	Barreto	1917	Africa (Algeria).
33. *Subulura travassosi*.	Barreto	1917	America (Brazil).
34. *Subulura lutzi*.	Barreto	1917	America (Brazil).
35. *Subulura trogoni*.	Barreto	1917	America (Brazil).
36. *Subulura bentocruzi*.	Barreto	1917	America (Brazil).
37. *Subulura carlosi*.	Barreto	1917	America (Brazil).
38. *Subulura olympioi*.	Barreto	1917	America (Brazil).
39. *Subulura protina*.	Baylis	1919	Africa (Uganda).

Revising the super-family *Ascaroidea* in 1913, HENRY and RAILLIET restablished the genus *Subulura* according to MOLIN; this opinion is generally accepted, and, latterly SUERAT tried to revive the genus *Allodapa* DIESING.

HALL shares the opinion of SEURAT.

In a note we published (vid. BARRETO 1917) we proved that the genus *Allodapa* cannot exist in helminthology, as the typical species of DIESING has all the characteristics of the genus *Subulura*. Those on which SEURAT and HALL based their classification of the formed genus i, cannot be left in the helminthological system as they are slight variations to be used only in distinguishing the different species of the same genus.

Diagnosis.

Subulurinae: Mouth dorso-ventral, hexagonal or elliptic of which the are with three small indistinct equal lips and cephalic papillae mostly. Buccal cavity distinct, furnished with three small teeth at the base; oesophagus always ending in bulb lateral. Membranes often found. *Male*: spicules, two equal or unequal, with accessory piece. Sucker spindleshaped or ellipsoid without chitinous rim. Caudal expansion rudimentary or wanting. Caudal papillae arranged in longitudinal rows, never more than eleven pairs. *Female*: Vulva only slighlly apparcnt, opening in the middle third of the body, duct turned either towards the anterior or posterior extremity. Uterus with diverging branches (?). Ova elliptic with thin shell, with or without embryo when laid.

Habitat: Ventricle and intestine (chiefly caecum) in birds; generally the large intestine in Mammals; also intestine of reptiles.

Type species: – *Subulura acutissima* MOLIN 1860.

List of species of the genus Subulura.

1. Subulura distans (Rud. 1809) Railliet & Henry, 1912.

Ascaris distans Rudolphi, 1809 *nec* Travassos, 1913 – p. 128 – 1913 a, p. 20 – 1913 b, p. 300, Pl. 29 fig. 23.
Ascaris distans Rudolphi, 1819 – p. 38.
Ascaris distans Dujardin, 1845 – p. 220
Ascaris distans Diesing, 1851 *pro parte* vol. 2, p. 146.
Heterakis distans Schneider, 1866 – p. 73, text-fig.; Pl. 3, fig. 10.
Heterakis distans Stossich, 1888 *pro parte* p. 291 (15) Pl. 8, figs. 32 and 38.
Subulura distans Railliet & Henry, 1913 p. 258.
Subulura distans Railliet & Henry, 1913 – p. 680.
Subulura distans Travassos, 1913 *pro parte* 1913 (a), p. 20, Pl. 3, fig. 23 1913, (b), p. 301, Pl. 29, fig. 23.
Subulura distans Gedoelst, 1916 – p. 41.
Habitat: Large intestine and caecum of:
Cercopithecus (C.) sabœus (*L*).
Cercopithecus (*C.*) *callithrichus* Geoff.
Cercopithecus (Rhinostictus) *cephus* L.
Cercocebus (*C.*) *fuliginosus* Geoff.
Cercocebus (C.) *collaris* Gray.
Cercopithecus sp.
C. patas Schreber.
C. brazzae Milne Edw.
Geographic distribution: Africa.

2. Subulura subulata (Rud., 1819) Railliet & Henry, 1913.

Ascaris subulata Rudolphi, 1819 – p. 38, 269 and 746.
? *Ascaris caprimulgi* Rudolphi, 1819 *n. nud.* (Cat. Mus. Vien. mss.) – p. 55.
Ascaris subulata Dujardin, 1845 – p. 169.
Ascaris subulata Diesing, 1851 *pro parte* vol. 2, p. 173.
Heterakis forciparia Schneider, 1866 *pro parte* – p. 75 text. fig.
Heterakis forciparia Stossich, 1888 *pro parte*—p. 288 (12). Pl. 7, fig. 25.
Ascaris subulata Parona, 1889 – p. 761.
Ascaris subulata Stossich, 1896 *pro parte* p. 70 (76).
Subulura subulata Railliet & Henry, 1913 p. 680.
Subulura strongylina Travassos, 1913 *pro parte* – 1913 (a) p. 18, 20 21 Pl. 3, fig. 22 – 1913 (b), p. 297 and 301. Pl. 29, fig. 22.
Subulura subulata Seurat, 1914—1914 (a) p. 157 – 1914 (b), p. 198.
Habitat: Intestine of:

Caprimulgus ruficollis Temm.
Caprimulgus europæus L.
Caprimulgus sp. (Engoulevent).
Geographic distribution: Spain (Algeciras); Corsica.

3. Subulura forcipata (Rud, 1819) Railliet & Henry, 1913.

Ascaris forcipata Rudolphi, 1819 *pro parte nec* Diesing, 1851, *nec* Seurat, 1914 – p. 657 and 746 – vol. 2, p. 147 – 1914 a, p. 157, text-fig. 4; 1914 b, pag. 201, text-figs. 1 – 3.
Ascaris forcipata Dujardin, 1845 *pro parte* p. 171.
Ascaris subulata Diesing, 1851 *pro parte* vol. 2 p. 173.
Heterakis forcipata Schneider, 1866 *pro parte* – p. 75, text-fig.
Heterakis forciparia Stossich, 1888 *pro parte* – p. 288 (12) Pl. 7, fig. 25.
Ascaris subulata Stossich, 1896 *pro parte* 70 (76).
Subulura strongylina Railliet & Henry, 1912 *pro parte*—p. 258.
Subulura forcipata Railliet & Henry, 1913 p. 680.
Subulura acutissima Railliet & Henry, 1913 *pro parte* – p. 676.
Subulura strongylina Travassos, 1913 *pro parte* – 1913 (a), p. 18, 20 and 21, Pl. 4 fig. 22 – 1913 (b), p. 297, and 301. Pl. 29 fig. 22.
Subulura acutissima Travassos, 1913 *pro parte* – 1913 (a) p. 18, 19 and 21. Pl. 4 figs. 26 27 – 1913 (b), p. 297, 298 e 303. Pl. 30, figs 26 and 27.
Subulura forcipata Seurat, 1914 *pro parte* 1914 a, p. 156. fig. 4 1914 (b), p. 201, text-fig. 1 – 3.
Habitat: Intestine and caecum of
Coccyzus melanocoryphus Vieill.
Coccyzus minor (Gm.).
Diplopterus nævius (L).
Piaya cayana (L).
Guira-guira (Gm)
Geographic distribution: Brazil.

4. Subulura strongylina (Rud, 1819) Railliet & Henry, 1912.

Ascaris strongylina Rudolphi, 1819 *nec* Cobbold, 1879 – p. 641 – p. 447.
Ascaris strongylina Dujardin, 1845 – p. 171.
Ascaris strongylina Diesing, 1851 – vol. 2, p. 150.
Heterakis forciparia Schneider, 1866 *pro parte* – p. 75 text-fig.
Heterakis forciparia Stossich, 1888 *pro parte* – p. 288 (12) Pl. 7, fig. 25.
Snbulura strongylina Railliet & Henry, 1912 *pro parte* – p. 258.
Subulura strongylina Travassos, 1913 *pro parte* – 1913 (a), p. 18, 20 and 21, Pl. 3, fig. 22 – 1914 (b), p. 297 and 301. Pl. 29, fig. 22.
Habitat: intestine of:
Crypturus tataupa Temm.
Crypturus sp.
Tinamus sp.
Odonthophorus capueira (Spix).
Geographic distribution: Brasil.

5. Subulura reclinata (Rud, 1819) Barreto 1917. (1)

Ascaris reclinata Rudolphi, 1819—p. 657 and 738.
Ascaris reclinata Diesing, 1851 – vol. 2, p. 174.
Ascaris reclinata Stossich, 1896 – p. 74 (68).
Habitat: Intestine of *Crotophaga ani* L. and *Crotophaga major* L.
Geographic distribution: Brazil.

6. Subulura allodapa (Creplin, 1853) Railliet & Henry, 1913.

Oxyuris allodapa Creplin, 1853 *nec* Seurat, 1914 – p. 59. – 1914 (a), p. 154, text-figs. 1 – 3 – 1914 b p. 196.
Allodapa typica Diesing, 1860 – p. 644.
Heterakis forciparia Schneider 1866 *pro parte* – p. 75 text-fig.

(1) – In this paper.

Heterakis forciparia Drasche, 1882 *pro parte.*
p. 120 Pl. 7 figs. 5 and 7.
Ascaris microdactyli Marcgrafi n. nud. coll. Mus. of Vienna.
Heterakis forciparia Stossich, 1888 *pro parte* – p. 280 (12) Pl. 7 fig. 55.
Heterakis suctoria Stossich 1888 *pro parte* p. 292 (16) Pl. 8, figs. 29 e 33
Heterakis suctoria Gendre, 1909 *pro parte* p. 8 and 10.
Subulura allodapa Railliet, & Henry, 1913 – p. 680.
Subulura suctoria Railliet, & Henry 1913 *pro parte* – p. 680.
Subulura strongylina Travassos, 1913 *pro parte* – 1913 a, p, 18, e 21 Pl. 3, figs 22 – 1913 b, p. 297 Pl, 29 figs 24 and 25.
Subulura suctoria Travassos, 1913 *pro parte* – 1913 (a), p. 19 e 21 Pl. 3, figs. 24 e 25 – 1913 (b), p. 298, and 302. Pl. 29, figs. 24 and 25.
Subulura allodapa Seurat, 1914 *pro parte* 1914 a, p. 154, text-fig. 1 – 3.
Allodapa allodapa Seurat 1914 *pro parte* 1914 p. p. 196.
Subulura allodapa Barreto, 1917 – 1917 p. 243.
Habitat: intestine and caecum of *Cariama cristata* L.
Geographic distribution: Brazil

7. Subulura jacchi (Marcel, 1857) (1) Railliet & Henry, 1913.

Ascaris distans Diesing, 1851 *pro parte* vol. 2, p. 146.
..... Marcel, 1857 – p. 340.
..... Leuckart, 1858 – p. 108.
Ascaris jacchi Diesing, 1860 – p. 667
Heterakis distans Stossich, 1888 *pro parte* p. 291 (15 Pl. 8, figs. 28 and 32.)
Ascaris jacchi Stossich, 1896 – p. 68 62.
Subulura jacchi Railliet & Henry, 1913 – p. 680.

(1) – *apud* Stossich 1896 p. 62.

Subulura distans Travassos, 1913 *pro parte* 1913 a, p. 20. Pl. 3, fig. 23 1913 b, p. 301, Pl. 29, fig. 23.
Habitat: intestine of
Callithrix jacchus L.
Callithrix chrysoleucus (Natt.)
Callithrix melanurus Geoff.
Midas (*M*) *bicolor* Spix.
Callicebus caligata Natt.
Geographic distribution: Brazil.

8. Subulura acutissima Molin, 1860.

Subulura acutissima Molin, 1860 – p. 332.
Subulura acutissima Diesing, 1860 – p. 644.
Subulura acutissima Drasche, 1882 – p. 120, Pl. 7, figs. 11 – 14.
Heterakis acutissima Stossich, 1888 – p. 289 (13) Pl. 8 fig. 30 and 34, Pl, 9, fig. 45.
Subulura acutissima Railliet & Henry, 1912 – p. 258.
Subulura acutissima Railliet & Henry, 1913 *pro parte* – p. 679.
Subulura acutissima Travassos, 1913 *pro parte* – 1913 (a), p. 19 and 21. Pl. 4, figs. 26 and 27 – 1913 (b), p. 297, 298. 299 and 303. Pl. 30, fig. 26 and 27.
Subulura acutissima Hall, 1916 – p. 52
Habitat: Intestine and proventricle of *Scops brasilianus* (G.)
Geographic distribution: Brazil.

9. Subulura (?) annulata (Molin, 1860), Travassos, 1913.

Heteracis annulata Molin, 1860 – p. 340.
Heteracis annulata Diesing, 1860 – p. 643.
Heterakis annulata Stossich, 1888 – p. 292 (16.)
Heterakis annulata Railliet & Henry 1912 p. 676.
Subulura annulata Travassos, 1913 – (a) p. 19 – 1913 (b), p. 298.
Habitat: intestine of
Xenodon severus (L).
Geographic distribution: Brazil.

10. Subulura suctoria (Molin, 1860) Raillet & Henry, 1912.

Ascaris forcipata Rudolphi, 1819 *pro parte* p. 660 e 746.
Ascaris forcipata Dujardin 1845 *pro parte* p. 171.
Ascaris subulata Diesing, 1851 *pro parte* vol. 2 p. 173.
Heteracis suctoria Molin, 1860 – p. 341.
Heterocis suctoria Diesing, 1860—p. 643.
Heterakis suctoria Schneider 1866 *pro parte* – p. 75 text-fig.
Heterakis suctoria Drasche 1882 *pro parte* p. 119 and 120 Pl. 7, figs 8 – 10.
Ascaris subulata Leidy 1885 – p. 10.
Heterakis forciparia Stossich, 1888 *pro parte* p. 288 (12) Pl. 7 fig. 25.
Heterakis suctoria Stossich 1888 *pro parte* p. 292 (16) Pl. 8 figs. 29 and 33.
Heterakis suctoria Gendre, 1909 *pro parte* p. 8 and 10 text-fig. 1 p. 11.
Heterokis suctoria Gendre 1911 *pro parte* p. 73.
Subulura suctoria Railliet & Henry, 1912 p. 258.
Subulura suctoria Railliet & Henry 1913 *pro parte* – p. 680.
Subulura strongylina Travassos, 1913 *pro parte* – 1913 (a), p. 18 – 21. Pl. 3, fig. 22 – 1913 (b), p. 297 299 and 301. Pl. 29 fig. 22.
Subulura suctoria Travassos, 1913 *pro parte* – 1913 (a), p. 19 and 21. Pl. 3, fig. 24 and 25 – 1913 (b), p. 298 299 and 302. Pl. 29 figs 24, 25.
Allodapa suctoria Seurat 1914 *pro parte* 1914 b, p. 196.
Habitat: intestine of
Caprimulgus sp. (Bacurão).
Caprimulgus rufus Bodd.
Caprimulgus nigrescens Cab.
Caprimulgus vociferus Wilson.
Podager nacunda Vieill. (Coruacão).
Stenopsis candicans Pelz.
Lurocalis semitorquatus (Gm.)
Hydropsalis climacocercus Tsch.
Nyctidromus albicollis (Gm.)
Nyctibius æthereus (Wied).
Nyctibius grandis (Gm.)
Nyctibius jamaicensis (Gm.)
Heliotreptus anomalus (Gould).
Geographic distribution: Brazil.

11. Subulura papillosa (Molin, 1860) Railliet & Henry, 1913.

Ascaris papillosa Molin, 1860 – p. 338.
Ascaris papillosa Diesing 1860 – p. 658
Ascaris papillosa Drasche, 1882 – p. 129. Pl. 9, fig, 24.
Heterakis papillosa Stossich, 1888 – p. 291 Pl. 7, fig. 24.
Subulura papillosa Railliet & Henry, 1912 p. 258.
Subulura papillosa Railliet & Henry, 1913 p. 680.
Subulura papillosa Travassos, 1913 – 1913 (a), p. 19 – 1913 (b), p. 298.
Habitat: intestine of
Cyanocorax cayanus (L).
Geographic distribution: Brazil.

12. Subulura (?) perarmata (Ratzel 1868) Railliet & Henry, 1913.

Heterakis perarmata Ratzel, 1868 – p. 150. Pl. 4, figs. 8 – 11.
Heterakis perarmata Stossich 1888 – p. 298 (13) Pl 9 fig. 44.
Subulura (?) perarmata Railliet & Henry, 1913 – p. 680.
Subulura perarmata Travassos, 1913 – 1913 (a), p. 19 – 1913 (b), p. 298.
Habitat: intestine of
Tarsius tarsius (Erxleb).
Geographic distribution: (?).

13. Subulura andersoni (Cobbold, 1876) Railliet & Henry, 1913.

Ascaris andersoni Cobbold, 1876 – p. 296. Pl. 21, figs. 14 and 15.
Ascaris andersoni Stossich, 1896 – p. 61 (67)
Subulura (?) andersoni Railliet & Henry 1913 – p. 680.
Subulura andersoni Hall, 1916 – p. 53, text-fig. 58.
Habitat: intestine of

Sciurus sp.
Geographic distribution: Asia (India).

14. Subulura curvata (Linstow, 1883) Railliet & Henry, 1913.

Heterakis curvata Linstow, 1883 – p. 291, Pl. 7 fig. 24.
Heterakis curvata Linstow, 1886 – p 15 fig. 24 (*apud* Stossich 1888 – p 287.)
Heterakis curvata Stossich, 1888 – p. 287 (11) Pl. 6, fig. 22.
Subulura curvata Railliet & Henry, 1913, p. 680.
Heterakis curvata Travassos, 1913 – 1913 (a), p. 8 – 1913 (b), p. 276.
Subulura curvata Seurat, 1914 – 1914 (a) p. 156 – 1914 (b), p. 196.
Habitat: Intestine of
Caccabis saxatilis var. *chukar* (Gray).
Geographic distribution: Asia (Turkestan)

15. Subulura otolicni (v. Beneden, 1890) Railliet & Henry, 1913.

Strongylus otolicni van Beneden, 1890 p. 389 figs. 1 – 7.
Subulura otolicni Railliet & Henry, 1913 - p. 680.
Subulura otolicni Gedoelst, 1916 – p. 43; text. fig. p. 44.
Habitat: intestine of:
Galago (G.) galago Schreber.
Galago (Hemigalago) demidoffi Fischer
Galago mossambicus Peters.
Geographic distribution: Africa (Guinea).

16. Subulura differens (Sonsino 1890) Railliet & Henry, 1913

Heterakis differens Sonsino, 1890 – p. 136.
Heterakis differens Railliet, 1895 – p. 409.
Heterakis suctoria Gendre, 1909 *pro parte* p. 8 and 10; text-fig. 2 (p. 11).
Heterakis suctoria Gendre, 1911, *pro parte* p. 73.
Subulura differens Railliet & Henry, 1912 p. 258.
Subulura differens Railliet & Henry, 1913 p. 680.
Subulura suctoria Railliet & Henry 1913 *pro parte* – p. 680.
Subulura differens Travassos, 1913 – 1913 (a), p. 19 1913 (b), p. 298.
Subulura strongylina Travassos 1913 *pro parte* – 1913 (a), p. 21 – 1913 (b), p. 299 and 302.
Allodapa suctoria Seurat, 1914 *pro parte* 1914 (b), p. 197.
Subulura suctoria Gedoelst, 1916 – p. 41, text-fig. p. 42.
Habitat: intestine of
Gallus domesticus L.
Numida meleagris L.
Francolinus bicalcaratus (L).
Geographic distribution: Cosmopolitan species; Europe: Italy (Pisa); Africa: Dahomey, Guinea (Fouta-Djalon) Algeria (Algiers), America: Brazil (Piauhy).

17. Subulura sarasinorum (Meyer 1896) Railliet & Henry, 1913.

Filaria sarasinorum Meyer, 1896 – 1896 b, p. 72 Pl. 4 figs. 10-15 Pl. 5 figs. 20-22.
Subulura sarasinorum Railliet & Henry, 1913 – p. 680.
Habitat: Intestine of
Loris gracilis Geoff.
Geographic distribution: Asia (India, Ceylon).

18. Subulura (?) gracilis (Linstow, 1890) Railliet & Henry, 1913.

Oxysoma gracilis Linstow, 1899 – p. 20 Pl. 5 figs. 64 and 65.
Subulura (?) gracilis Railliet & Henry, 1913 – p. 680.
Habitat: intestine of *Francolinus* sp.
Geographic distribution: Linstow obtained his material from the Zoological Garden in Berlin.

19. Subulura (?) acuticauda (Linstow, 1901) Railliet & Henry, 1913.

Oxysoma acuticauda Linstow 1901 – p. 415. Pl. 13 fig. 14.

Heterakis acuticauda Linstow 1909 – p. 448.
Subulura acuticauda Railliet & Henry, 1913 – p. 680.
Habitat: Intestine of
Numida rikwae Rchb.
Geographic distribution: Africa.

20. Subulura recurvata (Linstow, 1901) Railliet & Henry, 1913.

Heteracis recurvata Linstow, 1901 – p. 412. Pl. 13, fig. 7.
Subulura recurvata Railliet & Henry, 1913 p. 680.
Subulura recurvata Travassos, 1913, 1913 (a) p. 19 – 1913 (b) p. 298.
Heterakis recurvata Seurat, 1914 – 1914 (b) p. 201.
Habitat: intestine of
Eurystomus afer Gray.
Geographic distribution: Africa.

21. Subulura rimula (Linstow, 1903) Railliet & Henry, 1913.

Heterakis rimula Linstow, 1903 – p. 110. Pl. 5, fig. 5.
Subulura rimula Railliet & Henry, 1913- p. 680.
Subulura rimula Travassos, 1913. 1913 (a) p. 19 – 1913 (b) p. 298.
Habitat: Intestine of
Centropus sinensis (Steph.)
Geographic distribution: Asia (Siam).

22. Subulura rima (Linstow, 1906) Railliet & Henry, 1913.

Heterakis rima Linstow, 1906 – p. 252, Pl. 17, fig. 8.
Subulura rima Raillet & Henry 1913 – p. 680.
Subulura rima Travassos. 1913. 1913 (a) p. 19 – 1613 (b) p. 298.
Heterakis rima Seurat, 1914. 1914 (b) p. 202.
Habitat: caecum of:
Houbara undulata (Desf.)
(*Otis houbara;* Africa).
Houbara macqueenii (*nec* Desf.) (Bcht.)
(*Otis houbara;* Asia)
Geographic distribution: Linstow does not mention whether the bird from the Zoological Garden of Königsberg, was of asiatic or of african origin.

23. Subulura poculum (Linstow, 1909) Railliet & Henry, 1913.

Heterakis poculum Linstow, 1909 – p. 449 text-fig. 2.
Subulura poculum Railliet & Henry, 1913 p. 680.
Sobulura poculum Travassos, 1913. 1913 (a) p. 19 – 1913 (b) p. 298.
Heterakis poculum Seurat, 1914. 1914 (b), p. 197.
Habitat: intestine of
Francolinus adspersus Waterh.
Geographic distribution: Africa.

24. Subulura schebeni (Linstow, 1909) Railliet & Henry, 1913.

Heterakis schebeni Linstow, 1909 – p. 448, text fig. 1.
Subulura schebeni Railliet & Henry, 1913 p. 680.
Subulura schebeni Travassos, 1913. 1913 (a) p. 19 – 1913 (b) p. 298.
Habitat: intestine of
Cynictis penicillata (Cuv).
Geographic distribution: Africa.

25. Subulura leprincei (Gendre, 1909) Railliet & Henry, 1913.

Heterakis leprincei Gendre, 1909 – p. 7, text-fig. 1 and 8, p. 9.
Heterakis leprincei Gendre 1911 – p. 73.
Subulura leprincei Railliet & Henry, 1913 p. 680.
Subulura leprincei Travassos, 1913. 1913 (a) p. 19 – 1913 (b) p. 298.
Subulura leprincei Seurat 1914. 1914 (a) p. 156.
Allodapa leprincei Seurat 1914. 1914 (b) p. 197 text-figs. 4 and 5.
Habitat: caecum of
Microdipterix macrodipterus Afzel.
Caprimulgus fossii Hartl.

Caprimulgus ægyptius var. *saharae* Erlang.
Geographic distribution: Africa.

26. Subulura similis (Gendre, 1900) Railliet & Henry, 1913.

Heterakis similis Gendre, 1909 – p. 10, text-fig. 1 p. 11.
Heterakis similis Gendre, 1911 – p. 73.
Subulura similis Railliet & Henry, 1913 p. 680.
Subluura similis Travassos, 1913. 1913 (a) p. 19 – 1913 (b) p. 298.
Subulura similis Skrjabin.
Habitat: intestine of:
Coracias abyssinicus Bodd.
Eurystomus afer Gray.
Scops leucotis (Temm).
Centropus monachus Rupp
Centropus superciliosus.
Geographic distribution: Africa. (1)

27. Subulura elongata (Seurat, 1914) Barreto, 1917. (1)

Allodapa elongata Seurat, 1914. 1914 (c) p. 224 text-fig. 1 and 3 (p. 223).
Allodapa elongata Hall, 1916 – p. 60, text-figs 70 – 72 (p. 61 and 62).
Habitat: caecum of
Dipodilus campestris Levaill. (2).
Geographic distribution: Africa.

28. Subulura noctuœ (Seurat, 1914) Barreto, 1917. (2)

Habitat: intestine of
Carine noctua glaux Sav.
Geographic distribution: Africa.

Note 1. It seems to me that Gendre united several species under the same name of *Heterakis similis*, since the hosts mentioned by him belong to different orders and sub-orders. The first two birds are of the order *Picariae*, sub-order *Coraciae*; the third belongs to the order *Accipitres* sub-order *Striges*; the last to the order *Picariae* sub-ordre *Coccyges*. The material obtained from *Eurystomus afer* might be identified with *Subulura recurvata* (LINSTOW), found in a specimen of *Eurystomus afer*, from lake ny assa region but GENDRE separates the two species on account of the absence in LINSTOW's species of pepillae at the heihgt of the ucker.

Note (2) In this paper.

29. Subulura macronis (Stewart, 1914) Barreto, 1917. (2)

Heterakis macronis Stewart, 1914 – p. 165, 170 and 186, Pl. 19 figs. 17 to 24 Pl. 20 figs. 25 to 34.
Habitat: intestine of
Macrones aor Ham Buch.
Geographic distribution: Asia (India).

30. Subulura pigmentata Gedoelst, 1917 p. 153.

Habitat: intestine of
Sciurus prevosti Vigors and Horsf.
Geographic distribution: Asia (Sumatra)

31. Subulura seurati Barreto, 1917.

Subulura allodapa Seurat, 1914 *pro parte* 1914 (a) p. 154 text-figs. 1 - 3 p. 155.
Allodapa allodapa Seurat 1914 *pro parte* – 1914 (b) p. 196.
Subulura seurati Barreto, 1917. 1917 (a) p. 243.
Habitat: caecum of
Caccabis rufa (L).
Caccabis petrosa (Gm).
Geographic distribution: Africa.

32. Subulura halli, Barreto, 1917. (1)

Subulura forcipata Seurat. 1914 *pro parte* 1913 a, p. 156, text-fig 4 p. 155 – 1914 b, p. 201 text-fig. 1 – 3 (p. 199.)
Habitat: caecum of
Tetrax tetrax (L).
Geographic distribution: Africa.

33. Subulura travassosi n. sp.

Ascaris forcipata Rudolphi, 1819 *pro parte* p. 659 and 740.
Ascaris forcipata Dujardin, 1845 *pro parte* p. 171.
Ascaris forcipata Diesing, 1851 – vol. 2, p. 147.
Heterakis forcipata Schneider 1866 *pro parte* – p. 75 text-fig.

Note 1. In this paper.
Note (2) In this paper.

Heterakis forciparia Stossich 1888 *pro parte*—p. 289 (13). Pl. 7 fig. 25.
Subulura strongylina Railliet & Henry, 1912 *pro parte*—p. 258.
Subulura forcipata. Railliet & Henry 1913 *pro parte*— p. 680.
Subulura strongylina Travassos, 1913 *pro parte*—1913 (a), p. 21—1913 (b), p. 302.

Habitat: Intestine of
Malacoptila torquata (Ham u. Küst).
Chelidoptera tenebrosa (Pall),
Nonnula rubecula (Spix).
Monacha nigra (Müller).
Monacha morpheus (Hahn. u. Küst.)
Bucco collaris Lath.
Bucco macrorhynchus Gm.
Bucco tectus Bodd,
Bucco tamatia Gm.
Bucco striolatus Pelz.
Bucco rufiventris Natt.
Bucco swainsoni Gray and Mitsch.
Bucco chacuru Vieill.
Geographic distribution: Brazil.

34. Subulura lutzi n. sp.

Habitat: intestine of
Strix sp.
Geographic distribution: Brazil.

35. Subulura trogoni n. sp.

Habitat: intestine of
Trogon viridis L.
Geographic distribution: Brazil.

36. Subulura bentocruzi n. sp.

Habitat: intestine of
Trogon sp.
Trogon variegatus Spix.
Geographic distribution: Brazil.

37. Subulura carlosi n. sp.

Habitat: intestine of
Piaya cayana (L.)
Geographic distribution: Brazil.

38. Subulura olympioi n. sp.

Habitat: intestine of
Crypturus parvirostris Wagler.
Rhynchotus rufescens (Temm.).
Noctura maculosa (Temm.).
Geographic distribution: Brazil.

39. Subulura plotina Baylis, 1919.

Subulura plotina Baylis, 1919—p. 459, text-fig. p. 460.
Plotus rufus (Darter.)
Geographic distribution: Africa Uganda.

Description and species.

1. Subulura distans (Rudolphi, 1809) Railliet & Henry, 1913.

(Pl. 2 figs. 1 and 9).

Measurements:
♂ Length: 27 mm. (Duj).; 14,7 to 25,4 mm. (Dies.); 25 mm. (Schneider).
♀ Length: 40 mm. (DUJ).: 25,4 to 40,1 mm. DIES. Width 1,5 mm. DIES.

Body rather large, either of uniform width or thinning towards anterior extremity, spirally rolled. Head roundish, bare. Mouth triangular, with small lips leading directly to a pestleshaped oesophagus followed by spheric bulb.

Male: Slenderer than female. Tail curved like a hook, ending in a short point. Spicules unequal, very long and curved. Caudal bursa little developed. Genital papillae eleven in number. Elliptic sucker without chitinous ring.

Female: twice the size of male; straight; threadlike. Tail with diverging extremity. Eggs round.

Note.—The descriptions by Dujardin, Rudolphi, Diesing and Schneider are insufficient; we shall give all the characteristics cited by them. Some, like Diesing, Stossich and Travassos have counfounded this species with S. Jacchi.

Brazilian Species of the genus SUBULURA Molin, 1860

NAME OF SPECIES	AUTHOR	DATE	ORIGIN OF THE MATERIAL	COLLECTED BY	DATE OF COLLECTION
1. Subulura forcipata.	Rudolphi	1819	. . . . .	Natterer	1817—1835.
			Rio de Janeiro (Manguinhos).	Travassos	November the 28 th, 1913.
2. Subulura strongylina.	Rudolphi	1819	. . . . .	Natterer	1817—1835.
			Rio de Janeiro (Merity).	Travassos	1913.
3. Subulura reclinata.	Rudolphi	1819	. . . . .	Natterer	1817—1835.
			Pernambuco (Recife -Varzea).	Barreto	February the 3 rd 1916.
4. Subulura allodapa.	Creplin	1853	. . . . .	Burmeister	1851.
			Minas-Geraes (Lassance).	Theophilo	September—1916.
5. Subulura jacchi.	Marcel	1857	Amazonas (Barra do Rio Negro)	Natterer	1817—1835.
			Rio de Janeiro (Manguinhos).	Travassos	November the 28 th, 1914.
			Minas-Geraes (Lassance).	Theophilo	September 1916.
			Rio de Janeiro (Manguinhos).	Barreto	September the 6 th, 1917.
6. Subulura acutissima.	Molin	1860	. . . . .	Natterer	1817—1835.
7. Subulura annulata.	Molin	1860	Matto-Grosso (Caiçara).	Natterer	June the 20 th, 1826.
8. Subulura suctoria.	Molin	1860	Amazonas (Manáos).	Natterer	June the 19 th, 1834.
			Rio de Janeiro (Angra dos Reis).	Travassos	December 1913.
9. Subulura papillosa.	Molin	1860	Amazonas (Barra do Rio Negro)	Natterer	October the 2 th, 1830.
10. Subulura differens.	Sonsino	1890	Cosmopolitan species.		
			Piauhy (S. Raymundo).	Neiva	May the 7 th, 1912.
11. Subulura travassosi.	Barreto	1917	. . . . .	Natterer	1817—1835.
			Rio de Janeiro (Angra dos Reis).	Travassos	1913.
			Minas-Geraes (Lassance).	Theophilo	September 1916.
12. Subulura lutzi.	Barreto	1917	S. Paulo (Jacutinga).	Lutz	March—1907.
13. Subulura trogoni.	Barreto	1917	Rio de Janeiro (Angra dos Reis).	Travassos	December the 13 th, 1913
14. Subulura bentocruzi.	Barreto	1917	S. Paulo (Baurú).	Lutz	October 1906.
			Ceará (Rocha Museum).		
15. Subulura carlosi.	Barreto	1917	Rio de Janeiro (Angra dos Reis).	Travassos	1913.
			Rio de Janeiro (Ilha Grande).	Barreto	September the 29 th, 1916.
16. Subulara olympioi.	Barreto	1917	Minas-Geraes (Lagôas Periodicas de Nova-Granja).	O. Fonseca	June the 28 th, 1916.
			Minas-Geraes (Lassance).	Theophilo	September, 1916.

Habitat: harge intestine and caecum of
Cercopithecus (C) sabaeus (L).
Cercopthecus (C) callithricus Geoff.
Cercopithecus (Rhinostictus) cephus L.
Cercocebus (C) fuliginosus Geoff.
Cercocebus (C) Collaris Gray.
Cercopithecus sp.
Cercopithecus patas Schreber.
Cercopithecus brazzae Milne Edwards.
Geographic distribution: Africa.

2. Subulura subulata (Rudolphi, 1819) Railliet & Henry, 1913. (1)

Dimensions:
♂ Length 14.7 mm. to 16,8 mm. (Rud.) 7.5 mm. Seurat.

Note 1. We add the data of Seurat to the imprecise descriptio 1 by Rudolphi.

♀ Length: 18,9 mm. to 21 mm. (Rud.) 12 mm. Seurat; Width: 31 mm (Seurat).

Body slenderer at the extremities, especially at posterior one. Head bare, or with lateral expansions: lips small and indistinct.

Male: Tail very much curved. ending in a long fine point. Caudal expansions, very small or wanting. Sucker elliptic, elongate. Ten pairs of papillae, the first three united by a small cuticular expansion; three in the cloacal region; the tenth near the sucker. Spicules two, unequal, long and filiform with short point, measuring respectively 1,2 and 1,6 mm. Gubernaculum triangular, 0,11 mm. long. Anus 0,22 mm. from caudal extremity.

Female: Tail straight, conical, sharp-pointed 1/16 of the body. Oesophagus with bulb 1/8 of total length. Vulva slightly prominent, distinct, opening in front of the body 4,3 mm. from cephalic end. Oviduct remarkable on account of short sphincter which adheres to the vestibulum for nearly the whole of its length. Ova rounded, with embryo, longitudinal diameter 0,085 mm.; transversal diameter 0,056 mm.

Habitat: Intestine of
Caprimulgus ruficolis Temm.
Caprimulgus europeus L.
Caprimulgus sp. Engoulevent.

Geographic distribution: Spain (Algeciras), Corsica.

3. Subulura forcipata (Rudolphi, 1819) Railliet & Henry, 1913.

Plate XIV, figs. 1 & 2.

Dimensions:
♂ Length: 8,20 mm; width: 0,282 mm.
♀ Length: 13,6 mm.; width: 0,306 mm. measured at the height of the vulva.

Body, spindleshaped, yellowish white; the fore part spirally rolled towards the back Caudal part straight or turned ventrally in the female; in the male sometimes dorsally, at others ventrally. Cuticle very thin, transversally striated, the striae 0,036 mm one from the other. Lateral expansions small, wide, reaching the posterior limit of the bulb, 0,710 mm. long, nearly 0,074 mm. wide. Excretory pore a little beyond the nerve collar. Mouth with indistinct lips, with small papillae, placed laterally in two series, each with three papillae. Buccal cavity rather small, cylindro-conical, walls covered by a chitinous layer. Teeth, three, at the entrance of the oesophagus, lance-shaped, 0,030 mm. long. Oesophagus, with pear-shaped dilatation at the posterior extremity, the walls at first thin then thickening gradually until the terminal region; length 1,028 mm. Bulb spheroid, immediately behind the oesophagus; aproximate diameter 0,170 mm. Intestine dilated in the juxta-bulbous region, the last segment differentiated into a very short rectum.

Male: Tail funnel-shaped, curved now dorsally, now ventrally. Sucker small, elliptic, slightly prominent, ring without chitin; antero-posterior diameter 0,177 mm; in 0,592 distance from caudal extremity. Caudal expansions rudimentary. Ten pairs of papillae thus distributed: Three pre-anal, equal, ventral rather small, one on the level of the median part of sucker, one at the height of half the distance between sucker and anus, one at orifice and anus; two adanal, lateral, equal to foregoing; five post-anal, four of which are ventral that is, two large ones near the anus and two smaller ones near the caudal extremity; one lateral one between the two last mentioned groups. Spicules *equal*, slender, slightly curved, with sharp distal hook-like strongly curved extremity; length 0,677 mm. Straight gubernaculum extension 0,118. Anus 0,211 from caudal extremity.

Female: Posterior extremity sharp-pointed, at times rectilinear, at other times turned towards the ventral surface. Vulva small, slightly prominent, situated above the middle part of the body 5,654 mm. from the cephalic end. Oviduct short (1,285 mm, inclined towards the head of the nematode from the vulva onwards; vestibulum pear-shaped, with strongly chitinous lumen; neck inclined in obtuse angle towards the ventral surface 0,504 mm. long.; sphincter the same size as

vestibulum, chitinous, with a great number of cells in front of the horn, which is relatively small, 0,385 mm. with very muscular walls and internally but slightly chitinous layer. Uterus double, circumvolutions extending from the bulb to beyond the anus. Ova elliptical with exceedingly slender shell, and with the embryo completely formed when laid: length 0,048 mm., width 0,038 mm.

Development: Unknown.

Habitat: Intestine and caecum of
Coccyzus melanocaryphus Vieill.
Coccyzus minor Gm.
Diplopterus naevius L.
Piaya cayana L.
Guira-guira Gm.

Source: We obtained our material from the intestine of a *guira-guira* (Gm) in Manguinhos (Rio de Janeiro).

Material: From the Helminthological collection of the Instituto Oswaldo Cruz; nº. 1085.

Note 1. Under the denomination *Ascaris forcipata* Rudolphi describes various nematodes collected by Natterer in Brazil, in the intestines of birds belonging to the difierent groups *(Cuculus, Bucco* aud *Caprimulgus)*. Diesing in 1851 calls only the parasites of *Bucco forcipata*, as he considered the worms of *Cucculus* and *Caprimulgus* identical with *Ascaris subulata* Rud. which is a parasite of the European *Caprimulgus*.

Schneider gives a series of European and American birds as hosts of the *Heterakis forcipata* and, though he finds it strange that in so many birds of different genera there should be only one kind of parasite, with its own geographical distribution, he justifies his opinion from the fact that he did not find other varieties in the specimens he studied, but only a great difference in their dimensions.

After Schneider, nearly all the authors who studied this species included in the rubric *forcipata* the worms inhabiting birds of quite different habits which live n quite definite parts of the globe.

We are of opinion that it is more correct to separate the nematodes into different species according to the habits of their bird-hosts and their respective geogrephical distribution. Thus we designate *Subulura forcipata* the intestinal parasite of the Brazilian *Coccygiformes* of which the characteristics are identical with the above written diagnosis.

Note 2. We gather from the descriptions by Schneider of *Heterakis forcipata*, that he described specimeus of *Subulura suctoria*, found in *Caprimulgus*.

4. Subulura strongylina (Rudolphi, 1819) Railliet & Henry 1912. ([1] & [2])

Pl. II fig. 6: Pl. IV fig. 2: Pl. XI figs. 3 & 4.

Dimensions:
♂ Length: 4,36 to 12 mm. width: 0,308
♀ Length: 5,6 to 18,7 mm. width: 0,411 mm. at the level of the vulva.

Body white, filiform, sharpened at the extremities: cephalic part inclining towards the dorsal surface, as well in the male as in the female: caudal segment straight in the female, slightly ventrally curved in the male. Very slender cuticle, transversally striated, the striae being 0,0019 mm. distant one from the other. Lateral cephalic expansions well developed, stretching from the buccal orifice to the middle part of the bulb, striped transversally with numerous striae close together, with five or six very long longitudinal striae; length 1,367 mm. width 0,065 mm. Nerve collar situated between the cephalic extremity and the excretory pore. Excretory pore opening in the middle part of the oesophagus. Mouth in the form of an irregular hexagon, with badly defined lips, surrounded by six small papillae, disposed in two lateral rows of three, of which the middle ones are the largest. Buccal cavity cylindro-conical, narrow in the beginning and widening out at the back where it is twice as large as at the opening; thick inner layer of chitin; depth: 0,048 mm. Three almost rectan-

Note (1) The dimensions of the specimens obtained from the intestine of *Crypturus sp.* are somewhat variable a fact, which led us to believe that might possibly be a two distinct species. However, a lenghty examination of the material revealed an identical arrangement of the genital papillae, and the same length and shape of the spicules in the males; in the females: the same aspect of the vulva and, unchanging morphology of the ovijector and so we conclude that there is only one species which we identify with *Subulura strongylina*. Rud. As to the females, some are perfectly developed, and others not pregnant, although in a state of complete evolution, the vulva and different parts of the ovijector being distinctly visible.

Note (2) Perhaps the parasite of *Odontophorus capueira* (Spix) is not *Subulura strongylina*, as this host belongs to the order *Gallinae; Crypturus* belongs to the order *Crypturi*.

gular teeth at the entrance of the oesophagus, at the back of the buccal cavity, 0,019 mm. long. Oesophagus rectilinear, with pear-shaped expansion at the juxta-bulbous extremity; as to stucture it may be said to be divided into three parts: the first measuring about 0,076 mm. is but slightly muscular and its lumen is barely covered with chitin; the second is larger measuring 9,456 mm. and is more muscular and more thickly covered with chitin: the third about 0,532 large, has very muscular walls and forms the terminal expansion of the organ. Length of oesophagus: 1,040 mm. Spheroid bulb immediately after the oesophagus 0,205 in diameter, with three laminae forming an insosceles triangle inside. Intestine dilated in the juxta-bulbous section, entirely straight, ending in a short rectum with sleghtly thick wall.

Male: Excretory pore 0,465 mm. from the anterior limit of the body. Tail conical, slightly curved towards the ventral surface, with a straight, chitinous appendix measuring 0,102 mm. Sucker spindle-shaped, without chitinous rim; antero-posterior diameter 0,169 mm; posterior extremity 0,450 mm. from caudal extremity Genital expansions rudimentary. Eleven pairs of papillae distributed as follows: three preanal, ventral, equal and large ones: one at the side of the sucker, at the junction of the higher third and the lower two-thirds of this organ, one immediately above the anus, one equally distant from the lower limit of the sucker and of the cloacal orifice; two adanal equal ones: one lateral, the other ventral; six post-anal: of these four are ventral and two lateral; of the ventral ones two are larger, equi-distant and nearer to the anus, two lesser and situated near the posterior extremity of the lateral ones, one is large and at a level with the third post-anal ventral one, counting from the anus, the other is very small and between the third and fourth ventral ones. Spicules two, alike and extremely chitinous; with curved distal extremity; slightly sinuous with very sharp point, accompanied by an almost straight gubernaculum: length of spicules 1,180 mm; length of gubernaculum 0,169 mm. Anus 0,186 mm. from posterior extremity.

Female: Excretory pore 0,186 mm. from the cephalic extremity; tail straight aculate ending in a chitinous formation 0,102 mm. long. Vulva small, only very slightly prominent above the middle of the body. Oviduct, 0,950 mm. long, turned towards the cephalic extremity, its vestibulum sinuous, neck forming an acute angle with the ventral surface of the body; sphincter small. Horn of uterus equal to 2/3 of oviduct, muscular, turned suddenly towards the caudal extremity, seeming hook-like; afterwards it fuses with the uterus. Uterus bi-lateral with diverging ramifications; circumvolutions reaching to the first part of the intestine and to the anus. Eggs, elliptic, with thin shell, with embryo when laid; length 0,084 mm. width: 0.067. Anus 1,053 mm. from posterior extremity.

Development: Unknown.

Habitat: Intestine of

Crypturus tataupa Temm.
Crypturus sp.
Tinamus sp.
Odonthophorus capueira Spix.

We studied material from *Crypturus sp.*

Source: Rio de Janeiro (Merity).

Material: From the helminthological Collection of the Instituto Oswaldo Cruz, nos. 1080 and 1082.

5. Subulura reclinata (Rudolphi 1879) Barreto, 1917.

(Pl. 11 fig. 6).

Dimensions:

♂ Length: 11. mm.: width 0,359 mm.

♀ Length: 14.30 to 30,5 width: 0,514 mm. at the height of the vulva.

Body threadlike, white, with pointed extremities: the front part slightly dorsally deviated: the posterior one generally rectilinear in female, spirally curved in male. Cuticle thick, aparently in two zones, the inner one anhistous, smooth, the external one transversally striated, the striae 0,0021 mm. one from the other. Lateral cephalic expansions developed

in the middle, disappearing at the level of the initial part of the intestine; length: 1,590 mm. Nerve collar placed at the height of the junction of 1/3 of front part and 2/3 posterior part of oesophagus, 0,372 mm. from the cephalic limit. Excretory pore on ventral surface, beyond the nerve ring, more or less in middle zone of oesophagus. Mouth limited by three indistinct lips, furnished with six small papillae in two lateral series, each one with three papillae, the central one a little larger than the lateral ones. Buccal capsule small, may be divided into two parts; a higher sub-cylindrical one, narrowing in the middle part, with walls made of strong layers of chitin; a lower and ampler one, containing three small teeth of rectangular shape, of which the front sides may be rounded like the fingers of a glove: length: 0,016 mm. Oesophagus rectilinar, terminal segment dilated, pear-shaped: walls furnished with numerous transversal muscles and some longitudinal ones: length: 1,207. Bulb spheroid, joined to the oesophagus by a small canal, lodging three sub-triangular plates in the shape of an isosceles triangle, the sides of which are extremely chitinous: length 0,251 mm.; width: 0,211 mm. Intestine with slightly accentuated initial dilatation, rectilinear in its length, ending by a differentiation in the form of a rectum, at the end of which is the anus.

Male: Tail sharpening from the inferior limit of the sucker, decidedly curved towards the ventral surface, ending in on aculeiform formation 0,106 mm. in size. Sucker ellipsoid, without chitinous rim 0,143 mm. in length; the posterior end 0,503 mm. from the caudal extremity. Caudal expansions very much atrophied. Papillae eleven pairs, thus placed: three preanal, large, ventral: one at the side of the sucker at the junction of superior 1/3 and inferior, 2/3 one close to the anus, another at an equal distance from the anus and the sucker; two adanal, very large: one lateral, another sub-ventral: six post-anal ones of which two are larger and four ventral, equidistant the two larger near the anus, the two lesser ones near the caudal extremity; two lateral: a large one at the height of the third ventral one, counting from the anus and one between the two last post-anal ventral ones. Spicules *unequal* in length and width: the lesser one being more voluminous and about 2/3 of the length of the longer one: greatest length: 1,515 mm. width 0,019 mm; lesser one: length 1,105 mm.: width: 0.025 mm. Gubernaculum almost rectilinear with larger inner extremity; length: 0,160 mm. Anus 0,211 mm. from posterior extremity.

Female: Tail very sharp-pointed, ending in a chitinous appendix; 0,140 mm. in length. Vulva prominent, cleft-shaped, opening above the middle of the body. Oviduct long. (1,285 mm.); vestibulum relatively short, with neck turned at an almost rectangular angle, towards the ventral surface, with thick muscular walls; lumen limited by thin chitinous layer: sphincter small: horn very muscular, four times the length of the vestibulum. Uterus double, with hooks extending from the oesophagal bulb even beyond the anal orifice. Eggs elliptic, with very thin shell, with undeveloped embryo when laid; length: 0,076 mm. width: 0,050 mm. Anus 1,285 from posterior extremity.

Development: Unknown.

Habitat: Intestine of *Crotophaga ani* L. and *Crotophaga major* L.

Source: We described specimens from *Crotophaga ani* L. from Pernambuco (Recife-Varzea).

Material: Nos. 1,087 and 1,088 of the Helminthological collection of the Institute Oswaldo Cruz.

6. Subulura brachydapa (Creplin, 1853) Railliet & Henry, 1913.

Plate. III, Figs. 4 & 8; Plate. XII, Figs. 1 & 8.

Dimensions:

♂ Length: 7 to 10 mm.; width 0,33 mm.

♀ Length: 10 to 14 mm.; width 0,44 mm.

Body yellowish, straight in most of its length; anterior extremity like a truncated cone, generally inclined towards the dorsal surface; caudal extremity rectilinear and sharp-pointed in the female; ventrally curved like a fish-hook in the male, terminating in a cuti-

cular appendix in both sexes. Cuticle with very close transversal striae, 0,0021 mm. from each other, crossed by two lighter longitudinal striae. Lateral expansions narrow, lightly striated transversally, extending from the head to the middle part of the body in the male; in the female they occupy only the anterior third of body. Nerve ring at the junction of the posterior 4/5 and anterior 1/5 of the oesophagus. Excretory pore opening on the ventral surface, a little beyond the nerve ring, in the anterior third of the oesophagus. Mouth hexagonal, with indistinct lips, surrounded by six small papillae. Mouth capsule bottle-shaped, with walls covered by thick chitinous plates of charateristic aspect. (vid. fig. 2). Teeth, at the entrance of the oesophagus, three in number, with rounded outline. Oesophagus with posterior extremity dilated, club-shaped, united by a short duct to the pear-shaped bulb, containing triangular chitinous laminae, which continue directly on to the intestine. Intestine with larger initial part, rectilinear, slightly inclined towards the ventral surface and followed by a small rectum with light and thin walls.

Male: Nerve collar 0,338 mm. from cephalic end. Excretory pore 0,550 mm. from anterior extremity. Oesophagus: 1,285 mm. in length. Tail very sharply curved towards the ventral surface. Sucker witout ring, surrounded by radiating muscular fibres, 0,719 mm. from caudal extremity: length 0,169 mm. Genital expansions developed, beginning a little above the cloaca. Papillae, eleven pairs thus arranged: three pre-anal, ventral, large and equal ones: one in the middle part of the rim of the sucker, one at equal distances from the sucker and the cloaca, another immediately above the anus; two adanal: a larger lateral one, another smaller ventral one; six post-anal: three large ones at equal distances, the two first ventral and the third lateral, two small ventral ones near the caudal extremity, a very small ventral one between the above mentioned groups. Spicules slightly unequal in length, strongly chitinous, with the near extremity slightly dilated, the distal one sharppointed; length of larger spicule 1,525 mm; of lesser one: 0,465 mm; greatest width: 0,033 mm. Gubernaculum measuring 0,152 mm. Anus 0,169 mm. from posterior extremity.

Female: Nerve collar 0,337 from cephalic end. Excretory pore 0,485 mm, from the same. Oesophagus 1,336 mm. in size (without bulb). Tail straight, very sharp-pointed. Vulva slightly prominent, of irregular outlines, placed just in front of the middle part of the body. Oviduct turned to the cephalic extremity, measuring about 0,762 mm.; vestibulum relatively short (0,313) containing generally three eggs; when seen laterally showing the neck deviated towards the ventral plane of the body, with a good deal of chitinous covering: sphincter 0,211 mm. long with a group of large cells which are easily seen; Horn, measuring 0,348 mm., walls with thick muscular layers, becoming suddenly, slighter in the preuterine region. Anus 1,028 mm. distant from posterior extremity.

Development: Unknown.

Habitat: Intestine and caecum of *Cariama cristata* (L).

Source: Minas Geraes (Lassance.)

Material: N. 1039 to 1093 and 1121–1132 of the Helmintological Collection of the Institute Oswaldo Cruz.

7. Subulura jacchi (Marcel, 1857) Railliet & Henry, 1913.

Pl. IX Fig. 1 Pl. XIII Figs. 1 to 5).

Dimensions:

♂ Length: 10,2 to 14,2 mm.: width: 0,514 mm.

♀ Length: 13,4 to 17,9 mm, width: 0,616 mm.

Body of sanguinolent tint, rectilinear: anterior extremity in form of a truncate cone, straight or slightly deviated towards the dorsal surface; posterior part sharp-pointed, strongly curved ventrally in the male; straight in the female, ending in a short chitinous appendix. Cuticle of middle thickness, transversally striated, the striae 0,0029 mm. distant one from the other, crossed by a clearly seen longitudinal line. Lateral expansions developed, disappearing beyond the first part of the in-

testine; 0,067 mm. wide. Nerve collar situated approximately at the junction of the anterior and the posterior parts of the oesophagus. Excretory pore opening in the ventral surface, at the height of the middle part of the oesophagus. Mouth very small, hexagonal, surrounded by six small papillae arranged in two lateral series each with three papillae, the middle ones being the most voluminous Buccal cavity small, cylindro-conical with strong chitinous support, more dilated in the posterior part where are small sub-quadrangular teeth. Oesophagus rectilinear, with piriform dilatation in terminal part; united with spheroid bulb by a short canal. Intestine with dilated initial part, quite straight.

Male: Nerve collar 0,296 mm. from cephalic end. Anal orifice at 0,65 mm. Oesophagus: 0,925 mm. in length. Bulb about 0,254 mm. in diameter. Sucker cleft-like, ellipsoid without chitinous armature, surrounded by radiating muscular fibres, arranged in two groups one on each side of the organ; lower limit 0,71 mm. from caudal extremity; length 0,211 mm. Almost no caudal expansions. Genital papillae eieven pairs in number voluminous, thus arranged: three pre-anal ventral ones; the first very voluminous at the side of the upper third of sucker; the second large, half way between sucker and anus; the third slightly smaller above the ano-genital orifice; two ad-anal large and equal, one lateral, another ventral, six post-anal: four ventral; two larger ones near the anus, two much smaller ones near the caudal apendix, two lateral: one voluminous, one just above the second ventral group very small; its positiony varies from above the large lateral ones to below the penultimate ventral, one. Two spicules of slightly differing length, sinuous, slightly dilated in the proximal, sharply pointed in the distal extremity: 1,694 mm. in length; width 0,059 mm. Gubernaculum almost straight measuring 0,194 Anus 1,164 from caudal extremity.

Female: Nerve collar 0.338 mm. from head. Excretory pore 0,508 from same. Oesophagus: 1,156 mm. long. Diameter of bulb 0,282. Vulva slightly protruding, just above the middle of the body; vulvar orifice quadrangular. Ovijector going from vulva towards cephalic extremity measuring 0,899 mm. Vestibule generally containing three or four eggs, with thick muscular walls and slight chitinous lay3r 0,448 mm. long. Sphincter 0,169 mm. long. Horn with thin muscular layer, ample lumen, containing five or six eggs 0,398 long. Uterus with divergent branches; uterine hooklets surrounding the gut in its whole lenght. Ellipsoid eggs with thin shell containing embryos when laid, with the following diameters: 0,067 mm. and 0,050 mm. Anus 0,693 mm. from tail end.

Development: On examining faeces of *Callithrix jacchus* we found several specimens of *Subulura jacchi* and found not only numerous eggs containing perfectly developed embryos (vid. Plate XIV fig. 2) but also very lively larvae with the following caracteristics Body with very sharply pointed extremity: 0,512 mm. long. and 0,12 wide. Mouth round, circular, with small vestibulum, 0,012 mm. Digestive tube rectilinear, with ampulliform bulb, 0,177 mm. distant from cephalic end, 0,022 long and 0,006 wide. Anus 0,048 mm. from tail end (vid. Plate XIV, fig. 1). In order to follow the development of these larvae, we made culture media of them, mixing the faeces, diluted with water and animal charcoal finely pulverised and distributing the material in on slides. Some of the slides were exposed to the light, others we kept in darkness. We saw no change either in the eggs or in the morphology of the larvae during thirty-seven days observations. We only remarked that the activity of the larvae diminished progressively until it became null within eighteen days.

Habitat; Intestine of:

Callithrix jacchus L.
Callithrix chrysoleucus Natt.
Callithrix melanurus Geoff.
Midas (M) bicolor Spix.
Callicebus calligata Natt.

From Minas Geraes (Lassance) and Rio de Janeiro.

Material; Helminthological Collection of the Instituto Oswaldo Cruz. Nos. 1099 to 1108, 1133 to 1134.

8. Subulura acutissima Molin 1860. (1)

Plate IV, figs. 3, 5, 10 and 11.

Dimensions:

♂ Length; 7 mm. width: 0,30 mm.

♀ Length: 12 mm. width 0,30 mm.

Body filiform, sharpened at extremities, especially at the posterior end. Head roundish, with six papillae, two lateral and four submedian. Mouth hexagonal, small; at entrance of oesophagus three teeth of peculiar shape. Lateral expansions narrow.

Male: Tail tapering, sharp-pointed, strongly curved. Sucker large, fusiform, far from caudal extremity. Caudal expansions present. Spicules equal, long, large, spirally curved with accessory piece; genital papillae ten on each side, of these five post-anal and five pre-anal; the tenth papilla near the sucker.

Female: Tail very sharp-pointed, straight. Vulva in the posterior part of the body. (2) Anus far from posterior extremity.

Habitat: Intestine and proventricle of *Scops brasilianus* Gm.

Geographic distribution: Brazil.

9. Subulura annulata (Molin 1860) Travassos 1913. (3)

Dimensions:

♂ Length: 6 mm; width: 0,2 mm.

♀ Length: 8 mm. width: 0,2 mm.

Anterior extremity truncate. Mouth terminal, circular, large and bare. Lateral expansions linear.

Male: Tail sharp-pointed. Caudal expansions narrow. Seven genital expansions: five post-anal and two pre-anal. Sucker large and circular, distant from anus. Spicules long, filiform, curved, with thin point, distant from tail end.

Female: Tail ending in very thin point. Vulva prominent situated in anterior part of body. Anus distant from posterior extremity.

Habitat: Intestine of *Xenodon severus* L.

Geographic distribution: Brazil, Matto Grosso, Caiçara.

10. Subulura suctoria (Molin 1860) Railliet & Renry, 1912.

Plate 4 figs. 2, 7, and 9, Plate 12 figs. 1 and 2.

Dimensions:

♂ Length: 11,8 to 13,8 mm. width 0,359 mm.

♀ Length: 20 to 23 mm. width 2,539 mm. at the level of the vulva.

Body white, filiform, the anterior part spirally curved towards the dorsum; the caudal part generally rectilinear in the female, spiral in the male, the spiral being curved ventrally. Cuticle thin, transversally striated, the striations being 0,0024 mm. one from the other. Lateral cephalic expansions small, dwindling away before the middle part of oesophagus. Nerve collar at level of the junction of the anterior 1/4 with posterior 3/4 of oesophagus. Excretory pore opening in a slight depression of the cuticle, on ventral surface between nerve collar and middle of oesophagus. Mouth with indistinct lips, garnished with six small papillae distributed in two lateral rows, each made up of three papillae. Buccal cavity small, cylindrical, with walls protected by thick layers of chitin. Three triangular teeth, 0.025 mm. long. Oesophagus rectilinear, with pear-shaped dilatation at posterior extremity, moderately muscular, more considerably so at the level of terminal dilatation. Bulb piriform, connected at apex with oesophagus. Intestine without dilated first segment, directly con-

---

Note 1. Above, we give MOLIN'S description with the modifications introduced by DRASCHE.

Note 2. As to the vulva being situated in the posterior part of the body, we think it is a mistake of Molin's probably due to the faultiness of primitive technique; he himself says he cannot positively affirm that the point he saw was really the vulva, as he could not follow the direction of the ovijector through the body (vid, Molin-1860 page 333 (observations acompaning a description of *Subulura acutissima*).

Note 3. We mention the characteristics noticed by Molin; they are certainly incomplete and lack in precision we give them however only calling the organ which Molin gives as anus, a sucker, justifying this opinion by the situation of the genital opening and by the description of this organ by the above author.

nected with bulb, of a uniform diameter until near the anus, where it ends in a rather short thin walled rectum with spacious lumen.

Male: Buccal cavity: length: 0,050; width: 0,033 mm. Excretory pore at 0,420 mm. from cephalic extremity. Oesophagus: 1,185 long. longitudinal diameter of bulb 0,254 mm.; transversal diameter 0,203 mm. Posterior extremity funnel-shaped, ending in short rectilinear appendix. Sucker ellipsoid, without chitinous rim, 0,135 mm. long 0,296 mm. distant from genital pore. Caudal expansions very atrophied. Papillae: 11 pairs arranged thus: three pre-anal, ventral, voluminous, one at the side of the sucker, at the level of the of the juncton of the superior 1/3 with the lower 2/3 of the rim of that organ, one near the lower limit of sucker, one just above the anus; two ad-anal, large and equal: one lateral, the other ventral; six post-anal: five ventral and one lateral; of the ventral, two are voluminous and near the anus: two are lesser and near the posterior extremity: one is very small and between the two preceding groups: the large lateral one is situated on the level of this last one. Spicules *equal*, curved, with distal sharpened extremity at infundibiliform proximal end; length: 1,100 mm.; width 0,025 mm. Gubernaculum rectilinear and small (0,127 mm.) Anus 0,211 mm. from tail.

Female: Buccal cavity: length: 0,067 mm.; width: 0,042 mm. Excretory pore 0,508 mm. from anterior extremity. Oesophagus: length 1,285 mm. Transversal diameter of bulb: 0,205 mm.; antero-posterior one: 0,231 mm. Tail straight or slightly ventrally curved, aculeiform, ending in small appendix. Vulva small, not salient, cleft, in front of the middle part of body. Ovijector relatively short, turning towards cephalic extremity measuring 0,779 mm.; vestibule small (0,313) mm. slightly muscular internal chitinous layers; sphincter long, in comparison with the other species of, *Subulura*, 0,254 mm. with slightly chitinous lumen conlaining generally from 3 to 6 eggs; horn the same length as sphincter, with muscular layer only slightly developed making a violent curve just before it joins the uterus. Uterus dichotomized after a length of 1,1799 mm., branches diverging, hooklets stretching from bulb of oesophagus till beyond anus. Ovaries parallel in front of vulva; eggs elliptic with very thin shell; when emitted they contain embryos: length; 0,051 mm. width: 0,045 mm. Anus 1,146 mm. from posterior extremy.

Development: unknown

Habitat: intestine of

Caprimulgus sp. Bacurao.
Caprimulgus rufus Bodd.
Caprimulgus nigrescens Cab.
Caprimulgus vociferus Wilson.
Podager nacunda Viell. Corucão.
Stenopsis candicans Pelz.
Lurocalis semitorquatus Gm.
Hydropsalis climacocercus Tsch.
Nyctidromus albicollis Gm.
Nyctibius aethereus Wied.
Nyctibius grandis Gm.
Nyctibius jamaicensis Gm.
Heliotreptus anomalous Hould.

Source: We describe material from *Caprimulgus sp.* from Angra dos Reis, Rio de Janeiro.

Material: Helminthological Collection of the Instituto Oswaldo Cruz nos. 1063 and 1064.

11. Subura papillosa (Molin 1860) Railliet & Henry, 1913.

Plate IV, fig. 6.

Dimensions:

♂ Length: 12 mm.: 0,3 mm.

♀ Body with fine, transversal striations. Anterior extremity sharp-pointed. Head round. Mouth, small, oval, with two lateral papillae. Oesophagus muscular, joined to bulb which has chitinous plates in interior. Lateral expansions narrow.

Male: Tail sharp-pointed. Sucker elliptic, witout chitinous ring, with strong radiating muscles. Caudal bursa slightly developed.

Spicules unequal. Six pre-anal and five (?) (1) post-anal ones; the first three pre-anal papillae are distant from each other; the last three form a group; the sucker is situated between the second and third pre-anal papillae.

Female: unknown.

Habitat: Intestine of

Cyanocorax cayanus L.

Geographic distribution: Brazil (Amazonas, Barra do Rio Negro.

12. Subulura (?) perarmata (Ratzel 1868) Railliet & Henry, 1913.

Plate. II, figs. 1, 2, 3 & 4.

Dimensions:

♂ Length: 6 to 7 mm.; width 0,4 mm.

♀ Length: 8 to 10 mm.; width 0;4 mm.

Mouth followed by vestibule approximately 0,05 mm. deep, having inside an organ formed by six chitinous plates, three large and curved ones and three lesser ones. Oesophagus about 1/8 of length of body, sinuous, communicating with bulb of 0,15 in diameter, showing three, half-moon shaped, chitinous plates.

Male: Spicules two, long, symmetrical with anterior extremity dilated, and the posterior sharp-pointed, measuring 2,5 mm. of which 0,5 mm. protrude at cloaca. Accessory piece ending obtusely, the opposite extremity being dilated and 0,15 mm. long. Sucker represented by elliptic depression, with slightly raised borders of radiating muscles; 0,5 mm. from tail end.

Female: Vulva a little behind (?) middle of body.

Habitat: intestine of

Tarsius tarsius (Erxleb).

Geographical distribution: (?)

Note 1. We tried to complete the description by Molin, with the data Drasche. This latter author gives the number of six pre-anal papillae although his illustration only shows five and says perhaps the number of post-anal may be six or seven though the only male that he ow ruew had an incomplete caudal extremity.

13. Subulura (?) andersoni (Cobbold, 1876) Railliet & Henry 1913.

Plate IV., figs. 10 & 11.

Dimensions:

♂ Length: 12,7 mm.

♀ Length: 19 mm. width 1,916 mm.

Body tapering in anterior part and very sharp-pointed in posterior extremity, in both sexes. Head simples and bare.

Male: Tail with oval appendix. Two curved spicules with small expansions at distal extremity.

Female: Tail with indistinct point. Eggs sub-spherical, when emitted they have embryos are 50,8 micra to 63,5 micra long. Vulva ?

Habitat: intestine of

Sciurus sp.

Geographical distribution: Asia (Nortwest of India).

14. Subulura curvata (Linstow, 1883) Railliet & Henry, 1913.

Plate III, Fig. 4.

Dimensions:

♂ Length: 14 mm. width: 0,54 mm.

♀ Length: 12,5 mm.: 0,54 mm.

Body, tapering in anterior portion. Head roundish, with three small lips, each with small papilla. Oesophagus by round bulb 1/9,7 of whole length. Tail sharp-pointed.

Male: Tail 1/58,5 of whole body. Spicules measure 1,2 mm. and 0,9 Sucker slightly developed. Eleven papillae: two pre-anal, two ad-anal, seven post-anal, of which four are ventral and three lateral.

Female: Posterior extremity long and pointed, comprises 1/8 of length of body. Vulva a little beyond the middle of the body dividing it in two parts in the proportion of 14:13. Eggs 0,039 mm. long and 0,026 wide.

Habitat: intestine of

Caccabis saxatilis var. chukar (Gray).

Geographical distribution; Asia (Turkestan).

15. Subulura otolicini (van Beneden 1890) Railliet & Henry, 1913. (1).

Plate V. figs. 3, 4, 5, 6. 7, 8, and 9.

Dimensions:

♂ Length: 8 mm.; width 0,4 mm.

♀ Length: 12,5 to 16,6 mm.; width: 0,64 to 9,65 mm.

Body cylindroid, stretched at extremities more at the back than the front, whitish. On the tegument very fine, transversal striations. Head slightly dilated and rounded in the front: at the apex a round capacious mouth, 35 to 45 micra in diameter; lips not distinct, but surrounded by six cephalic papillae; two lateral which are more voluminous and four sub-median ones; the mouth leads to a buccal cavity or vestibule 56 micra deep and 64 micra wide; in this are three chitinous teeth round the entrance to oesophagus. The latter measures 1,2 to 1,3 in length and is club-shaped; the posterior part is so dilated that its diameter is about three times that of the anterior part and forms a bulb with triturating apparatus which is in continuity with the rest of the oesophagus. Nerve collar surrounding the oesophagus a little behind a fourth of the anterior part.

Male: Dimensions: 8 mm. length, greatest width 0,4 mm. Oesophagus as long as 2/13 of whole length. Cloaca opening at 255 micra from tail end; tail end with two slightly developed lateral expansions; tail short, conic, lengthened by a 95 micra long appendix. In front of the cloaca, at a distance of about 480 micra, is a sucker with lengthened cleft, not bordered with chitin and measuring more or less, 160 micra. The ventral surface has 11 pairs of papillae, of which 3 pre-anal pairs the first being situated by the side of the anterior extremity of pre-anal sucker. The two spicules are equal, measuring about 1,8 mm. in lenghth and 28 micra in thickness; the anterior extremity slightly widenened; the posterior extremity ending in a point; spicules transversally striated in entire length accompanied by an accessory piece or gubernaculum, which measures 225 micra in length; with edges curved like gutters ending in a point behind; the genital tube extends to 3,3 mm. of anterior extremity.

Female: Dimensions 12,5 to 16,6 mm. in length, greatest thickness 0,64 to 0,65 mm. Oesophagus a little more than 110 of whole length. The anal orifice 1,4 mm. from tail end, tail 1/11 of the length of the body, long, conic, ending in sharp point, like that of the male. Vulva not salient, opening in the middle of the body, which it divides in the proportion of 7:9 coils of the genital tubes extending from 200–270 micra behind the bulb of the oesophagus to 500 behind the anus. The eggs are sub-globulose or ellipsoid and measure about 66×50 micra; shell fine, smooth; when emitted the eggs contain embryos.

Habitat: intestine of

Galago (G) galago Schreber.

Galago Hemigalago demidoffi Fischer.

Galago mossambicus Peters.

Geographical distribution; Africa (Guinea Kikondja).

16. Subulura differens (Sonsino, 1890) Railliet & Henry, 1913.

Plate II, figs. 8, Plate 8, fig. 5.

Dimensions:

♂ Length: 8,6 mm.; width; 0,282 mm.

♀ Length: 11,3 to 12,5 mm; width; 0,334 mm. at the height of vulva.

Body rectilinear, dirty yellowish colour; some specimens almost black, perhaps on account of the action of the preserving fluid. Anterior part conical, slightly deviatied towards the dorsal surface; caudal part gradually tapering, ending in a small chitinous appendix, straight in the female, spirally curved in the male. Cuticle thin, with fine transversal striations in the superficial layer. Lateral expansions well developed, extending from cephalic extremity to the anterior 1/5 of intestine; greatest width 0,022 mm. Nerve collar on the anterior third part of oesophagus. Excretory pore on ventral surface, a litle beyond the nerve collar, before the middle

Note 1. We transcribe the description by Godoelst.

part of oesophagus. Mouth with indistinct lips and small cephalic, equal papillae. Buccal cavity regularly cylindrical in anterior part, of which the walls are protected by thick chitinous plates, more dilated in the posterior segment where are to be found three teeth in from of an equiteral triangle. Oesophagus; rectilinear, club-shaped more dilated at posterior extremity, followed by sub-spherical bulb. Intestine dilated in the first part.

Male: Buccal cavity: 0,041 mm. long: 0,022 mm. wide. Oesophagus: 0,925 mm. in length. Bulb: antero-posterior diameter 0,231 mm; transversal diameter: 0,205 mm. Sucker ellipsoid, without chitinous ring, surrounded by radiating muscles, measuring 0169 mm. and with posterior limit 0,508 mm from tail end. Caudal expansions rudimentary. Ten pairs of papillae thus distributed: three preanal larger and ventral; one by the side of sucker at the height of its middle part, one at the level of the junction of the anterior 1/3 and the posterior 2/3 of the distance from sucker to anus; two ad-anal, large, lateral; five post-anal ones four ventral ones: two large ones at the cloacal orifice; two smaller ones near the tail end, one lateral, one between the ventral groups. Spicules equal and strongly chitinised; proximal extremity funnel-shaped, distal, sharp-pointed: length: 1,016; mm. width: 0,022 mm. Gubernaculum slightly curved, 0,101 mm. Anus opening in slight ridge 0,169 mm. from posterior extremity.

Female: Buccal cavity 0,054 mm. deep and 0,029 mm. wide. Oesophagus: 1,105 mm long. Bulb: 0,257 mm. and 0,231 mm. in diameter. Excretory pore 0,456 mm. from cephalic end, Vulva not salient, opening in a slight depression of cuticle above the midle of body, 5,397 mm. from head. Ovijector 0,677 mm. long, turned towards the anterior extremity; vestibule very long (0,338 mm.) with wide neck slightly curved, thin walls, and slightly chitinised lining. Horn muscular connected with vestibulum by small sphincter. Uterus double, uterine hooklets going round the intestine in its whole length but not passing the rectum. Ova almost spheroid, with very thin shell, containing embryos when laid 0,059 mm. in diameter longitudinally and 0,040 mm. transversally. Anus 0,771 mm from tail end.

Development: unknown

Habitat: intestine of

Gallus domesticus L.

Numida meleagris L.

Francelinus bicalcaratus L.

Geographical distribuition: Cosmopolitan species: Europe: Italy (Pisa); Africa: Dahomey, Guinea (Fouta-Djalon), Algeria (Algiers), Kiva: America: Brazil (Piauhy.)

Material: From *Gallus domesticus* found in S. Raymundo, Piauhy; in the helmintholo-gical collection of the Instituto Oswaldo Cruz n. 1086.

17. Subulura sarasinorum (Meyer, 1896) Railliet & Henry, 1913.

(Plate VI. figs 1 to 9.)

Dimensions:

♂ Length: 7,5 mm.; width; 0,046 to 0,52 mm.

♀ Length: 10 to 11,24 mm.; width: 0,57 to 0,7 mm.

Body yellowsh-white. Cuticle smooth, ringless, finely striated transversally. Median lines normally developed, lateral lines more considerable. Polymiarian muscle cells; muscular sections formed by fifteen elements. Ample lateral expansions, dwindling at the the posterior extremity of the oesophagus 1,4 mm. in length, i. e. about 1/6 of the body and 0.08 mm. of the greatest width. Excretory pore opening near the cephalic extremity, in median ventral line. Nerve collar situated in anterior part of oesophagus, in fron-of excretory pore. Mouth almost circular, in central position, slightly ventral with four badly outlined teeth and four large papillae. Buccal cnvity 0,057 mm. in length and 0,025 mm. in width. Oesophagus sinuous, with thick muscular walls, and internal lining, strongly chitinous, with bulb in which are three chitinous plates; length of oesophagus 1,4 mm. or 1/6 of body.

Male: Tail straight, sharp-pointed, beginning at anus. Caudal bursa regularly developed, beginning at level of the rectum; greatest

width, 0,027 mm. (1/6 of the body) at the height of anus. Ten pairs of genital papillae: four pre-anal and six post-anal, the later being the smaller. Two equal spicules, 2,5 mm. long and 0,016 mm. wide. Anus near posterior extremity, 25 mm. long.

Female: Tail ending in slender point. Vulva circular, exactly above the middle of the body. Ovijector, very large, sinuous, curved backwards' with muscular layer formed by circular and longitudinal fibres, first smooth afterwards irregular. Uterus continuing ovijector, divided into two branches reaching to oesophagus in front and anus behind. Eggs with comparatively thick shell, 0,081 mm. in longitudinal and 0,065 mm. in transversaly diameter, oval, containing embryos.

Habitat: intestine of *Loris gracilis* Geoff.

Geographical distribution: Asia, Southern and East India.

18. Subulura (?) gracilis (Linstow, 1899) Railliet & Henry, 1913.

Plate III figs. 1 & 8.

Dimensions:

♂ Length: 7,9 mm.; width: 0,3 mm.

♀ Length: 9,48 mm.; width 0,39 mm.

Cephalic end furnished with two small papillae. Buccal cavity with strongly chitinised walls. Oesophagus followed by bulb, spherical with inside (?). Tail with long and sharp point in both sexes.

Male: Oesophagus 1/8 of length of body. Tail 1/40. Spicules curved, 0,59 mm. in length, with strong border of orbicular muscles. Nine caudal papillae: three pre-anal and six post-anal, one pre-anal and one post-anal, lateral ones.

Female: Oesophagus equal in length to 1/9,3 of whole length. Tail 1/8. Vulva dividing the body in two in proportion the 11:13. Ova with embryos, 0,057 in length and 0,044 in width.

Habitat: intestine of *Francolinus sp.*

Geographical distribution: Linstow's material came from the Zoological garden in Berlin.

19. Subulura acuticauda (Linstow, 1901) Railliet & Henry, 1913.

Plate III fig. 7.

Dimensions:

♂ Length: 10,5 mm.; width: 0,39 mm

♀ Length: 14,8 mm.; width: 0,51 mm.

Cephalic extremity rounded. Oesophagus with bulb at end, corresponding in the male to 1/10,7, in the female to 1/12,5 of the whole length. Initial part of intestine dilated, posterior extremity sharp-pointed in both sexes, in the male equal to 1/38, in the female 1/18,7 of the length of the body.

Male: Spicules equal, measuring 0,58 mm. Sucker cleft-like, without chitinous border. Papillae eight, of which three pre-anal and five post-anal.

Female: Vulva situated above the middle of body, which it divides in two parts in the proportion of 11/14. Eggs 0,047 mm. long and 0,034 mm. wide.

Habitat: intestine of *Numida rikwae* Rchb.

Geographical distribution: West Africa Usanga Lake Rukwae.

20. Subulura recurvata (Linstow, 1901) Railliet & Henry, 1913.

Plate III fig. 6.

Dimensions:

♂ Length: 6,78 mm.; width 0,23 mm.

♀ Length: 9,26 mm. width; 0,43 mm.

Cuticle smooth. Cephalic extremity rounded, curved dorsally, with six papillae arranged in a circle. Caudal extremity sharp-pointed. Oesophagus followed by spheroid bulb equal to 1/7 in male, to 1/7,9 of whole length in female.

Male: Tail curved ventrally, equal to 1/25,7 of body. Spicules long, voluminous measuring 0,88 mm. Sucker elliptic, without horny border, flanked by bundles of diverging muscles. Nine caudal papillae (1) three pre-anal and six post-anal ones.

Female: Tail sharp-pointed, equal to 1/14

Note 1. LINSTOW only mentions 8 papillae but gives nine in corresponding illustration.

part of whole length. Vulva situated above the middle of body, dividing it into two parts, in the proportion of 11:15. Eggs numerous, 0,049 long and 0,036 wide.

Habitat; intestine of *Eurystomus afer* Gray.

Geographical distribution: East Africa Lake Nyassa.

21. Subulura rimula (Linstow, 1903) Railliet & Henry, 1913.

Plate III, fig. 9.

Dimensions:

♂ Length: 8,6 mm.; width: 0,34 mm.

♀ Length: 12 mm.; width: 0,58 mm.

Cuticle transversally ringed. Cephalic extremity obtuse, without lips or papillae. Tail end conical and sharp-pointed. Oesophagus ending in spherical bulb, measuring 1/7 of whole length in male and 1/8 in female.

Male: Tail equal to 1/27 of length of body. Testicle leaving only the first fifth of body free. Spicules 0,88 mm. Ten papillae: three pre-anal and seven post-anal ones. Sucker elliptic, between the first and second pre-anal papillae, surrounded by radiating muscles.

Female: Tale occupying 1/20 of body. Vulva at the junction of anterior third with two posterior thirds. Eggs almost round 0,049 mm. long and 0,041 wide.

Habitat: *Centropus sinensis* (Steph).

Geographical distribution: Asia (Siam.)

22. Subulura rima (Linstow, 1906) Railliet & Henry, 1913.

Plate III fig. 8.

Dimensions:

♂ Length 8,2 mm.; width: 0,40 mm.

♀ Length 9,3 mm.; width: 0,43 mm.

Cuticle with widely separated transversal rings. Mouth surrounded by six papillae: four large ones, one on each longitudinal line, and two lesser ones, one to the right and one to the left. Buccal cavity deep, leading to oesophagus, at the opening of which are three nodules, each with a conical tooth. Oesophagus followed by bulb; in the male equal to 1/5,3 and in the female to 1/5 of whole length. Lateral expansions cephalic.

Male: Tail 1/21 of the length of body. Spicules, the right-hand one equal to 0,48 mm., the left 0,70 mm. Accessory piece. Sucker elliptical, situated very much to the front. Nine papillae: one by the side of sucker, three pre-anal and five post-anal ones.

Female: Posterior extremity long, ending in a point, equal to 1/6,7 of total length. Vulva situated in front of middle of body, separating it in two parts, in the proportion of 51:53. Eggs not developed.

Habitat: caecum of

Houbara undulata (Desf).

Outis houbara from Africa.

Houbara macqueenii nec (Desf). (Becht).

Outis houbara from Asia.

Geographical distribution: LINSTOW, studied the material from the Koenigsberg Zoological Garden, not mentioning whether the host were Asian or African.

23. Subulura poculum (Linstow, 1909) Railliet & Henry, 1913.

(Plate III fig. 5).

Dimensions:

♂ Length: 4,5 mm.; width: 0,28 mm.

♀ Length: 8,5 mm.; width: 0,43 mm.

Cuticle with fine trasversal annelation. Lateral expansions cephalic. (?). Mouth surrounded by six papillae. Buccal capsule flattened. Oesophagus ending in bulb, represents 1/5,3, in male and 1/8,5 in female of total bodily length. Nerve collar behind buccal cavity. Excretory pore in front of oesophagean bulb.

Male: Tail equal to 1/29 part of body. Spicules long, slender-pointed, 0,79 mm, in length. Ten papillae: two pre-anal, two ad-anal, six post-anal. Sucker oval, surrounded by radiating muscles. Testicle reaching to initial part of intestine.

Female: Tail conical and sharp-ponited equal to 1/9,7 of length of body. Vulva in front of the middle of the body, separating it in two parts in the proportion 20:33. Genital organs stretch from beginning of oeso-

phagus to anus. Eggs with embryos 0,065 mm. long and 0,049 mm. wide.

Habitat: intestine of *Francolinus adspersus* Waterh.

Geographical distribution: German Southwest Africa.

24. Subulura schebeni (Linstow, 1909) Railliet & Henry, 1913.

(Plate III fig. 2).

Dimensions:

♂ Length: 5,8 mm.; width: 0,32 mm.

♀ Length: 7,3 mm.; width 0,32 mm.

Cuticle showing transversal rings. Cephalic extremity rounded. Mouth circular, leading to flat buccal cavity, with two lateral plates of chitin. Oesophagus with round and thickened posterior extremity equal to 1/5 in the male, to 1/6 of total length in the female. Excretory pore very much to the front, dividing the oesophagus in the proportions 4:9. Nerve collar dividing the same organ in the proportions 1:4.

Male: Tail equal to 1/37 of body. Spicules equal, sharp-pointed measuring 0,53 mm. Ten papillae: two pre-anal, three ad-anal and five post-anal ones, the penultimate lateral; and the last one double. Sucker ellipsoid.

Female: Tail ending in a point, represents 1/15 of total length. Vulva dividing the body in two proportional parts, 3:4. Genital organs stretching from end of oesophagus to anus. Eggs with embryos, with fine shell, 0,070 mm. long and 0,047 mm. wide.

Habitat: intestine of *Gynictis penicillata* (Cuv.)

Geographical distribution: German. Southwest (?) Africa

25. Subulura leprincei (Gendre, 1909) Railliet & Henry 1913.

(Plate VIII figs. 1 to 8; Plate X figs. 3 & 4.)

Dimensions:

♂ Length: 18 mm; width: 0,42 (Gendre.)
Length: 10,5 mm.; width: 0,30 mm. (Seurat).

♀ Length: 18,2 to 35 mm; width: 0,60 mm. (Gendre).
Length: 21 mm; width: 0,40 mm. (Seurat).

Body yellowish-white, tapering at extremities, especially at posterior end in both sexes. Cuticle finely striated. Lateral expansions narrow, stretching from head to near tail. Cephalic extremity rounded; without lips, or boundary line to separate it from the rest of the body. Mouth wide and circular, surroun by six equidistant papillae; four sub-median and two lateral ones. Buccal cavity cylindrical, with chitinous teeth at the bottom. Pharynx short and muscular. Oesophagus in the beginning narrower than pharynx, gradually thickening towards the posterior extremity where it is dilated and communicates with bulb, by means of a narrow pedicule. Intestine uniformely cylindrical, straight to the anus. Excretory pore situated on ventral surface, at the level of the anterior 1/4 of oesophagus.

Male: Oesophagus corresponding to 1/9,3 of total length. Tail 1/48 of body. Posterior region conical, sharp-pointed, ventrally curved with caudal bursa well-developed. Sucker oval, fibrous, far from cloaca. Thirteen papillae on each side: seven pre-anal and six post-anal; the post-anal are submedian, free, arranged in longitudinal rows, with the exception of two lateral ones: one pre-anal at the level of anus, another post-anal in the middle part of tail; of the pre-anal group, the two first ones are one above, one below the sucker; the two last at the level of the cloaca; of these, one is immediately above the anus; in the group of post-anal ones, the fourth is very small. Spicules two, equal, curved, delicately transversally striated, measuring 1,40 mm. (1 mm. SEURAT) presenting two distinct parts: the shortest ending in funnel-shaped dilatation is cylindrical; the other ending in sharp point is voluminous, prismatic and has a depression on the ventral surface. Accessoy piece triangular and hollowed, measuring 0,130 mm., according to SEURAT.

Female: Oesophagus measuring 1/15 of length of body; tail 1/25 (1200 mm. SEURAT) Straight conical tail, sharper-pointed and slen-

derer than that of male. Vulva indistinct, transversal, situated on ventral surface in the anterior half of body, (about 1/2,5 of body), (2,5 of the length). Ovijector very long, remarkable for globulous middle part of the sphincter, where the eggs are found just before they are ready to be emitted. (SEURAT) Uterus double, hooklets not reaching the anal region. Eggs roundish, 0,060 mm. long, and 0,051 mm. wide, with reticulated thick shell, composed of small polygonal plates in their first stadium, getting smoother and thinner as segmentation progresses and the embryos develop.

Habitat: caecum of

Microdipterix macrodipterus (Afzel).
Caprimulgus fossii (Hartl).
Caprimulgus aegyptius var v. *saharae* (Erlang).

Geographical distribution: Africa: French Guinea, Dahomey, Southern Tunis.

26. Subulura similis (Gendre, 1909) Railliet & Henry 1913.

(Plate II, fig. 7).

This species was incompletely described by GENDRE, who thought it presented great likeness to *Heterakis suctoria* MOLIN from which it differs only in the peculiar arrangement of the two penultimate caudal papillae of the male.

GENDRE does not mention any other characteristic of his *Heterakis similis*, only remarking that the vulva opens in front of the middle of the body about 1/2,3 of total length.

This author gives a list of several hosts of this species and calls attention to the different sizes of the same in the diferent individuals found in different birds. The following are his observations:

Coracias abyssinicus (Bodd).
♂ Length: 9,50 mm.; width: 0,40 mm.
Eurystomus afer (Gray).
♂ Length: 13,08 mm.; width: 0,40 mm.
♀ Length: 18,80 mm.; width: 0,40 mm.
Scops leucotis (Temm).
♂ Length: 12,60 to 15 mm. width: 0,30 to 0,636 mm.
♀ Length; 20,12 to 22,45 mm.: width 0,42 to 0,50 mm.
Centropus monachus (Rupp).
♂ Length: 0,4 to 13,08 mm.
♀ Length: 11,06 to 18,4 mm.

It seems to us that GENDRE put different species together, as the hosts to which he refers belong to distinct orders and suborders. Thus for instance, the first two birds belong to the order *Picariae*, sub-order *Coraciae;* the third to the order *Accipitres* sub-order *Striges;* the last to the order *Picariae*, sub-order *Cocciges*. The material from *Eurystomus afer* might be identified with *Subulura recurvata* LINSTOW, taken from an *Eurystomus afer*, from the region of Lake Nyassa; it was however GENDRE himself who separated it on account of the absence in LINSTOW'S species of the papillae which are present at the level of the sucker in *Subulura similis*.

SKRJABIN mentions a nematode found by him in *Centropus superciliosus*, which he classifies as *Subulura suctoria* MOLIN without describing it.

Taking into consideration that the material came from British East Africa, and that the host is *Centropus*, we resolved to identify it provisorily with *Subulura similis* from *Centropus monachus* from Dahomey (Africa).

Geographical distribution: Africa: French Guinea, Dahomey British East Africa.

27. Subulura elongata (Seurat 1914) Barreto, 1917. (1)

(Plate IX, figs. 1, 2 & 3).

Dimensions:

♂ Length: 16 to 20,5 mm.
♀ Length: 29 to 36 mm.; width: 0,65 mm.

Body slight. Cephalic alae narrow, delicately transversally striated, not going beyond the middle of oesophagus. Mouth surrounded by six papillae; buccal cavity distinctly divided into two parts: the upper part bordered

Note 1. In the present paper.

by a cuticular membrane, which is very thick; the lower part showing a thick cuticle and three coneiform sharp and slightly deviated points.

Male: Oesophagus very short, its length with bulb being 1/14 of that of body. Tail short. Sucker slightly developed, long, without chitinous ring. Caudal alae narrow. Ten pairs of caudal papillae, five of which are post-anal; the tenth pair slightly asymetrical. Caudal glands opening on the ventral surface between second and third pairs of papillae. Spicules equal, short, 0,250 mm. long i. e. 1/24 part of body. Gubernaculum in the shape of an arrow, 0,12 mm. long. Anus 0,23 mm. from tail end.

Female: Oesophagus very short, 1/17 of whole length including the bulb. Tail short, conical, measuring 0,915 mm. ; caudal glands opening in posterior 1/4 of tail. Vulva but slightly salient, situated before the middle of body, which it divides in the proportions 5:8. Ovijector bent backwards: horn very long, attaining 8 mm. Uteri stretch from 1 mm. behind the bulb to the beginning of rectum. Ovaries parallel in pre-vulvar region. Numerous eggs with embryos when ripe, 0,070 mm to 0,077 mm. long. and 0,053 to 0,065 mm. wide.

Habitat: caecum of *Dipodillus campestris* Levaill.

Geographical distribution: Africa: South Algeria: Bou-Saãda.

28. Subulura noctuae (Seurat, 1912) Barreto, 1917. (1)

(Plate 8 fig. 4).

Dimensions:

♂ Length: 14,5 mm.; width: 0,25 mm.

♀ Length: 22 mm.; width: 0,38 mm.

Body slender; oesophagus short, as tail also. Cephalic expansions reaching to oesophagean bulb. Buccal cavity divided into two distinct parts, with three small rounded teeth at base.

Note 1. In present paper.

Male: Oesophagus together with bulb equal to 1/10 of body. Tail small, with relatively long point (0,085 mm). Sucker small, like a longitudinal cleft. Caudal expansions well developed. Ten pairs of papillae, of which five are post-anal; of the pre-anal, ones, three are near the cloaca and the last near the sucker; caudal glands open on ventral surface between the second and third papillae. Spicules equal, filiform, 0,7 mm. in length. Gubernaculum long, triangular, prolongated in the front by two small ridges. Anus 0,3mm. from caudal extremity.

Female: Oesophagus short, measuring 1/14,5 of total length. Tail short (0,30 mm.). Caudal glands in posterior third. Vulva situated a little in front of the middle of the body, in the 3/7 of length. Ovijector running backwards; vestibulum short; horn very large, 5 mm. Uteri parallel, reaching from bulb to anus. Ovaries blackish, in second part of the body, behind the vulva.

Very numerous eggs, with embryos in the ripe state, measuring 0,050 mm. by 0,040.

Habitat: intestine of *Carine noctua glaux,* Sav.

Geographical distribution: Africa. Algeria, Bou-Saãda.

29. Subulura (?) macronis (Stewart 1914) Barreto (2)

Plate VI, fig. 10 to 17; Plate VII, fig. 1 to 10

Dimensions:

♂ Length 3,65 – 7,412 – 7,31 – 7,5 mm.; width 0,148 mm.

♀ Length: 6,596 — 7,82 – 8,5 mm. width: 0,129 – 0,1406 – 0,153 mm.

Body filiform, the anterior extremity tapering abruptly and curved dorsally; the posterior part gradually sharpening in the male,

Note 2. For the present we include this nematode in the genus *Subulura* on account of the appearence of the sucker without chitinous border. The absence of oesophagean bulb and accessory piece, as well as the situation of the vulva in the posterior part of the body, suggest that the nem. tode cannot be included in this genus. Stewart describes it as *Heterakis Acheilostomi* (Railliet), insinuating that it ought to be included with the *Subulurae*.

curved towards ventral surface. Cuticle smooth. Lateral lines 0,238 mm. wide, at level of oesophagus. Head rounded, without lips, with small cephalic alae. Mouth formed by a funnel-shaped shallow groove, at the anterior extremity of oesophagus, surrounded by narrow border of cuticle. This rim is thicker in the ventral than in the dorsal segment; the anterior end of the oesophagus is more prominent in the ventral than in the dorsal part, so that the transversal plane of the mouth is bent towards the dorsal surface. Oesophagus club-shaped, with dark pigmentation behind the nerve collar, has no bulb, and is 0,47 mm. to 0,85 mm. long. Nerve collar 0,31 to 0,42 mm. from cephalic end. Lateral expansions all the way down the body from head to near anus; transversal section of same like right-angle triangle; the greatest width 0,048 to 0,066 mm.; at the height of the posterior part of oesophagus. At the distance of 0,56 mm, from the head, is a narrow belt of protoplasm beginning at the lateral line, traversing the corresponding lateral ala to its edge; this is doubtless a sensitive organ.

Male: Tail measuring 0,44 mm, with curved dorsum ending in a caudal appendix, 0,073 mm. Genital bursa well developed, furnished with 24 papillae, disposed in five series: two sub-lateral, two sub-ventral at the level of the caudal expansions, one in in the space between the two expansions. The sub-lateral rows are formed by three post-anal papillae: one at the posterior limit of the first subventral papillae (the papillae are counted beginning at the caudal extremity), one near the second sub-ventral, one in the space betwen the second and third sub-ventral ones. The sub-ventral series consist of eight papillae: three post-anal, one ad-anal, and four preanal; of the post-anal the first very voluminous, near the posterior extremity, the second smaller, just above the afore-mentioned one, the third small just below the anus; the preanal: one above the anal orifice, one between the anus and the sucker, the fourth 0,14 mm. above the sucker. The median series consists of two papillae: one just above the anus, the other at the end of the lower canal. Sucker without chitinous rim, slightly prominent 0,45 mm. from caudal extremity. Spikes two, slightly chitinized, curved like the latter, without accessory piece, 0,765 mm. long. Anus 0,17 to 0,18 from posterior extremity.

Female: Tail conical and sharp-pointed. Vulva cleft-like, occupying 1/3 of total circumference, situated at the junction of the middle third of body with posterior. Anus 0,22 to 0,25 mm. from caudal extremity. Eggs 0,051 to 0,062 mm. long, ano 0,037 mm wide.

Habitat: intestine of *Macrones aor*. Ham Buch.

Geographical distribution: Asia (India).

30. Subulura pigmentata Gedoelst, 1917.

Dimensions:

♂ Length: 16,5 – 18,6 mm.: width: 640 to 750 micra.

♀ Length: 27 – 27,6 mm.; width; 0,7 mm.

Body whitish, cylindrical, more slender at extremities: cephalic extremity obtuse, round: caudal extremity conical, sharp. Teguments striated transversally, striations 3,3 to 4 micra distant from one another; Cephalic alae beginning a little behind the head, stretching to the posterior part of the oesophagus, which measures 1,35 to 1,9 mm. and is from 50 to 90 micra wide; the striations are transversal.

Mouth terminal, hexagonal, with great dorso-ventral diameter, contained by an ondulated border, individualising 6 small prominent lips, arranged by threes on each side of the great diameter; furnished with 6 well developed papillae; followed by buccal cavity or vestibulum, slenderer in the anterior part and larger in the posterior part enclosing the tridentate apparatus characteristic of the *Subulurae*. The oesophagus may be divided into two parts; the first larger slightly dilated part gets narrower at the anterior 1/4 or 1/5 and then widens into club-shape; the second part is separated from the first by a constriction, constituting the sub-globulous oesophagean bulb, containing the teeth. Nerve collar at the height of 1/4 or 1/5 anterior part

of oesophagus. Excretory pore opening a little behind the nerve collar. Intestine large in the initial part, narrowing towards the anus.

Male: Oesophagus equal to 1/9 to 1/11 of the body in length. Tail 1/51 part of body, curved like a fish-hook, ending in a very sharp point. Sucker cleft-like, without chitinous ring, 200 micra long, 1,5 mm. from anus.

18 pairs of caudal papillae: two in front of sucker: two at the same height as sucker: six between sucker and cloaca: three adanal one of these being lateral, and five post-anal of which one is lateral, and one single one median above the cloacal orifice. The last pre-anal, the two ventral ad-anal and the two first post-anal support each a spartially developed expansion on side. Spicules *equal*, without alae, transversally striated, the anterior extremity dilated, the posterior sharp-pointed: length 1,9 mm.; gubernaculum, 295 micra. Genital tube begins a little behind the anterior extremity of the spicules, goes straight forwards and becomes hook-like 3,9 mm. behind oesophagean bulb.

Female: Oesophagus about 1/12 of the body in length. Tail straight, conical, sharp-pointed, about 1/20 of body. Vulva opening in middle of body, which it divides in the proportions 2: 3. Ovijector turned backwards, relatively short, with cylindrical vestibulum, measuring 640 micra in length, and 140 micra in width, followed by a horn which opens obliquely by means of a valvule protected by a sphincter. Horn short, 410 micra long and 125 micra wide. Uterus single, 10 mm. in length, with hooklets turned backwards in the first part, which is 2,25 mm. in length and 200 micra in width: single in the beginning, afterwards divided into two parallel reservoirs; greatest diameter 200 micra, full of eggs.

Uterine hooklets stretch forwards until 320 micra from the bulb and backwards to 960 micra from tail. Eggs sub-globulous, 72—89 micra in length and 50—64 micra in width; the medium being 75—61 micra.

Habitat: intestine of *Sciurus prevosti raffflesi* (Vigors & Horsfield).

Geographical distribution: Sumatra.

31. Subulura seurati Barreto, 1917.

Plate IX figs. 1, 2 and 5.

Dimensions:

♂ Length: 14,5 mm. width: 0,49 0,50 mm.

♀ Length: 12,5 mm.; width: 0,585 mm.

Body of sanguinolent colour, thick, very tapering at posterior extremity, with two lateral expansions in the cephalic and oesophagean regions; lateral spots very apparent on account of their dark colour. Mouth hexagonal, drawn-out, with great dorso-ventral axis surrounded by six papillae. Buccal cavity with three small teeth at the base. Oesophagus dilated in the posterior region, continuing by clearly distinct bulb, with denticular apparatus. Nerve collar at the anterior third of oesophagus.

Male: Cloaca 0,43 mm. distant from caudal extremity. Sucker elliptic, drawn-out length ways, without chitinous ring. Caudal expansions narrow. Eleven pairs of papillae, of which five pre-anal; three of these adanal and the last at the side of the sucker. Spicules unequal, proportions 3-2, measuring respectively 1,35 mm. and 0,85 mm. Gubernaculum triangular 0,15 in length.

Female: Tail conical, comparatively short, 1,14 mm. long. Vulva situated in anterior region of body at 1/3 of its length. Ovjector remarkably large, more than 5 mm. Vestibulum piriform, bent towards the front, with thick internal cuticle, containing a small number of eggs, at most three. The sphincter is so united to the vestibulum as to allow of the free passage of the eggs; the walls of the sphincter are so elastic that reverse passage is impossible. Sphincter very long, and divided into three parts; the initial region remarkably muscular and thickly clothed with cuticle; the middle part globulous, charaterised by a thick external and internal secretory layer, becomes highly coloured with methylene blue; in this part the eggs remain some time; the last part is distinguished by a thin

internal layer of longitudinal muscular cells lined with thin cuticular membranes with longitudinal folds. Horn musculo-epithelial, very large: 4 mm. in the initial region; the epithelial cells in contact with the internal surface obscure the lumen of the canal; towards the front the horn becomes larger, the high and narrow epithelial cells being substituted by large flat cells confining a large space full of eggs with embryos. The ovijetor curves forwards, the horn curves backwards and joins the uteri which reach down to the posterior extremity of the body, and then rise to the oesophagean region, where they continue with oviducts and ovaries side by side in front of the vulva. Eggs very numerous, thick-shelled, 0,055 mm. by 9,045 mm.

Habitat: caecum of *Caccabis rufa* (L) and *Caccabis petrosa* Gm. (1)

Geographical distribution: Africa, Bou-Saãda, Algeria.

32. Subulura halli Barreto, 1917.
(Plate XI, figs, 1, 2, 3 e 4.)

♂ Length: 10,5 to 12 mm.; width: 0,54 mm.

♀ Length: 13,4 to 16 mm.; width: 0,6 mm.

Male: Teeth cuneiform, sharp-pointed. Oesophagus with bulb, equal to 1/6,5 of body. Tail, tapering, ending in a fine point, often ventrally curved. Cloaca 0,5 mm. from posterior extremity. Caudal expansions narrow. Eleven pairs of pedunculated papillae, five of which are pre-anal; the eleventh pair at the side of the sucker. Sucker 0,7 mm. from cloacal orifice. Spicules two, equal very long, (5 mm.) at the free end, with wing-like expansions, which are transversally striated, and which widen at the sub-terminal part. Gubernaculum is 0,12 mm. long.

Female: Body turned like a bow, the concave part being bent towards the dorsal surface, somewhat narrower at the anterior extremity, ending in a long, tapering, transparent tail, pointed at the end and corresponding to 1/8,5 of total length. Oesophagus including bulb, equal to 1/7,5. Vulva in front of the middle part of body, in the anterior 2/5 of the body. Ovijector with very short sphincter; horn first bent forwards, then suddenly bent backwards. Uteri reaching from the beginning of oesophagean bulb until 0,5 mm. from anus. Eggs 0,080 mm. long and 0,060 mm. wide.

Habitat: caecum of *Tetrax tetrax* L.

Geographical distribution: Africa, Maison-Carrée, Algeria.

33. Subulura travassosi Barreto, 1918.
(Plate XVI, figs. 1 to 5.)

Dimensions:

The dimensions of this species vary in the specimens we obtained from *Bucco swainsoni* and *Bucco chacuru*; this difference may however be due to the poor state of the material from *Bucco swainsoni*, in which the worms were out of shape and very shrunk.

Bucco swainsoni.

♂ Length: 3,0 to 4,6 mm. width 0,230 to 0,300 mm.

♀ Length: 6,0 mm.; width: 0,410 mm.

Bucco chacuru:

♂ Length: 5,60 to 7,70 mm.; width: 0,360 mm.

♀ Length: 7,70 to 10 mm.: width: 0,400 mm.

Body whitish, with tapering extremities, the anterior part generally straight, sometimes dorsally bent; posterior part sharp-pointed, ending in an appendix, ventrally curved in the male, straight in the female. Smooth cuticle with fine transversal striations (0,001 distant from one another) traversed longitudinal by two light lines and one dark line between the former. Lateral cephalic expansions very narrow (0,033 mm. wide), disappearing at the height of the bulb. Nerve collar in the anterior half of oesophagus. Excretory pore opening on ventral surface, a little beyond nerve collar. Mouth ellipsoid, with indistinct lips and six little papillae. Buccal ca-

Note 1. This species was found by Seurat in the caecum of the red partridge in North Africa; the author does not give the scientific name of its host. In the Catalogue of the Britsh Museum we find that the above nomenclature corresponds to the African red partridge.

vity cylindro-conical, shallow (0,042 mm), well-protected by strong chitinous plates, containing three lanceolate teeth. Oesophagus rectilinear, the posterior part being club-shaped with muscular walls communicating with the intestine by means of a spherical bulb which contains three stong chitinous plates. Intestinal tube rectilinear, initial portion dilated.

Male: Nerve collar 0,254 mm. from anterior extremity. Excretory pore 0,381 from same. Oesophagus: 1028 mm. long. Bulb, 0,231 mm. in diameter. Sucker small, shallow, ellipsoid, 0,385 mm. long, 0,508 mm. from caudal extremity. Genital expansions only slightly developed. Eleven pairs of papillae: three ventral, pre-anal, voluminous; one by the side of the middle third of the border of sucker, one just above the anus; another a little above the later, two ad-anal large ones: one lateral, one ventral below the last-mentioned; six post-anal: two large ventral ones near the anus: two smaller ones also ventral, near the tail end, one large lateral, one between these last-mentioned; one very small one at the root of the lateral one. Spicules two, *equal* strongly chitinised, the proximal extremity dilated and the distal end very sharp; length 1,710 mm. Curvilinear accessory piece 0,211 mm. large. Anus, 0,127 mm. from tail end.

Female: Nerve collar 0,254 mm. from head. Excretory pore 423 mm. from head. Oesophagus 1,156 mm. long. Bulb's diameter 0,254. Vulva cleft-like, not prominent, opening a little above the middle of body; ovijector 0,771 mm. long, starting from vulva, bent towards the cephalic extremity. Vestibulum 0,406 mm. in size, corresponds to half the total length of ovijector, and original in shape, (fig. 1); its lumen lined by thick layer of chitin, with thin and muscular walls. Sphincter very short, measuring 0,169 mm. Horn very short, with well developed muscular layer the thickness of which diminishes rapidly in the pre-uterine segment, continuing with the thin epithelium of the uteri: length 0,296 mm. Uteri, dichotomized, the diverging uterine circumvolutions reaching the first part of the intestine, and rarely passing the anus. Ovaries in opposite directions in front of the vulva. Eggs elliptical, with thin smooth shells, containing embryos when ripe and having the following dimensions: 0,077 mm. and 0,058 mm. in diameter. Anus 0,693 mm. from posterior extremity.

Life History: unknown.

Habitat: intestine of:

Malcoptila torquata (Ham. u. Kuest.)
Chelidoptera tenebrosa (Pall).
Nonnula rubecula (Spix).
Monacha nigra (Mueller).
Monacha morpheus (Hahn. u. Kuest.)
Bucco collaris (Lath).
Bucco macrorhyncus (Gm).
Bucco tectus (Bodd).
Bucco tamatia (Gm).
Bucco striolatus (Pelz).
Bucco rufiventris (Natt).
Bucco swainsoni (Gray & Mitsch).
Bucco chacuru (Vieill).

From Rio de Janeiro (Angra dos Reis) &, Minas Geraes (Lassance).

Material: Helminthological collection of the Instituto Oswaldo Cruz, nos. 1071 to 1075. (1)

34. Subulura lutzi Barreto, 1918.

(Plate XVII, figs. 1 to 4.)

Dimensions;

♂ Length: 14 mm.; width: 0,37 mm.

♀ Length: 14 to 22,5 mm.; width: 0,46 mm. at the height of vulva.

Body, white, rectilinear, filiform, with tapering and curved extremities, in opposite directions in the male. Cuticule fine transversally striated, the striations 0,024 mm. one from the other. The anterior part of body cone-like, with truncated apex, curved

Note 1. We attribute the differences we noticed, to the poor state of the material from *Bucco swainsoni* noticeable when compared with that from *B. chacuru*. The general aspect of the tail in males and the position of certain genital papillae, is not the same in the specimens from these two hosts. The only way to decide finally whether the parasites harboured by these *Bucconidae* are the same would be to obtain well preserved specimens from *Bucco swainsoni*.

towards the dorsal surface; this curve is stronger in the males, in some specimens of which the head is almost at a right angle to the body. Lateral expansions comparatively short, dwindling down at the posterior extremity of the oesophagus, delicately transversally striated; 1,272 mm. long. Excretory pore opening on the ventral surface a little beyond the nerve collar. Mouth ellipsoid, with indistinct lips, and six papillae, three on each side, the middle one larger than the two lateral ones. Buccal capsule small, conical, narrowing rather in the middle part, 0,055 mm. deep, 0,026 mm. wide in the widest part, and 0,035 mm. at the extremitites. Three small, equal, clubbed teeth at the extremity of buccal cavity, at the entrance to oesophagus, 0,034 mm. long. Oesophagus rectilinear, with pear-shaped posterior extremity, 1,24 to 1,60 mm. long, the walls muscular in the initial part, diminish gradually, thickening again after the middle part towards the posterior limit, being the thickest at the height of the terminal dilatation. The lumen of the oesophagus reveals chitinous layers, which thicken until the juxta-bulbular extremity. Bulb almost spherical, united to the oesophagus by a small canal of muscular walls, containing a thin layer of chitin; diameter of bulb, 0,25 mm.; bulbous apparatus formed by three sub-triangular plates with chitinous borders. Intestine dilated in the initial part, rectilinear at the terminal segment, with a thin-walled rectum.

Male: Excretory pore 0,558 mm. from cephalic extremity. Caudal part strongly curved towards the ventral surface, ending in an aculeiform appendix, 0,279 mm. long. Sucker ellipsoid, with chitinous border from front to back 0,213 mm.; its lower border 0,798 mm, from tail end. Almost no caudal expansions. Genital papillae eleven in number, arranged thus: three pre-anal ventral, one, the largest of all, at the level of the middle third of the sucker, one large one midway between the posterior limit of sucker and anus, one like the latter one in size near the anus; two ad-anal large: one sub-ventral, one on the lateral surface of body: six post-anal: two larger ones ventral, equidistant near the ano-genital orifice, two smaller ventral ones: one near the caudal extremity where the aculeiform appendix is inserted; one large, lateral one at the level of the second papilla counting from posterior extremity; finally one very small lateral one between the two last ventral papillae. Two *unequal* spicules distal extremity sharp-pointed; the larger one is thinner 1,270 mm. long. the lesser one sinous more strongly chitinised is 3/4 of the larger one and is 0,794 long. Gubernaculum slightly curved, with the concave side turned to the ventral surface; length 0,144 mm. Anus 0,169 from posterior extremity.

Female: Excretory pore 0,646 mm. from anterior extremity. Tail rectilinear, sometimes slighty turned towards the ventral surface, followed by appendix, like that of male, 0,110 mm. long. Vulva very prominent, with irregular rim, opening in the midle of the body, more or less at the junction of the anterior 1,3 and the posterior 2/3. Ovijector long, turned towards the cephalic end, that is it forwards from the vulva and then bends backwards; it measures 156 mm; vestibulum short (0,089 mm.), retort-shaped, with the neck abruptly curved at right angles. Sphincter small. Horn very long. (0,899 mm.). Uterus double, uterine hook-lets reaching from the first part of the intestine to a litle bey ond anus. Eggs elliptical, thin-shelled, containing embryos when laid, 0,033 mm. long and 0,055 mm. wide. Anus 0,443 mm. from posterior extremity.

Life History: unknown.

Habitat: intestine of *Strix* sp.

Origin São Paulo (Jacutinga).

Material: Helminthological Collection of the Instituto Oswaldo Cruz nos. 1070, 1119 and 1120.

35. Subulura trogoni n. sp.
(Plate XV, figs. 3 to 4).

Dimensions:

♂ Length: 7,4 mm.; width: 0,572 mm.
♀ Length: 15,5 mm.; width: 0,858 mm. at the height of vulva.

Body, white, rectilinear in the female

specimens; the ends curved in opposite directions in males. Cuticle thick, the superficial layer furrowed by shallow transversal striations, 0,0018 mm. one from the other; cuticle 0,015 mm. thick. Lateral cephalic expansions slightly developed, reaching to the initial part of intestine; length 0,223 mm.; width 0,055 mm. Nerve collar 0,344 mm. from cephalic extremity. Excretory pore opening through a slight groove of cuticle, in the ventral surface of the body, beyond nerve collar. Mouth with slightly accentuated lips, surrounded by six equal, small papillae. Buccal capsule shaped like the segment of a cone, larger in the posterior part, its walls with a covering of chitin of a peculiar form, length 0,045 mm.; width: 0,038 mm. Teeth three, subrectangular at the base of buccal capsule; length: 0,026 mm. Oesophagus rectilinear, dilated in the posterior part; moderately muscular. Bulb subspherical; transversal diameter larger than the antero-posterior one, containing strongly chitinised plates; length: 0,279 mm.; width: 0,353 mm. Intestine dilated like a calix; the first part, in which the bulb penetrates has a small rectum in the terminal segment, 0,232 mm. long.

Male: Excretory pore 0,620 mm. from anterior excremity. Tail tapering, ventrally curved, with formation of aculeiform chitin rectilinear 0,093 mm. Sucker small, ellipsoid, without horny rim, 0,423 mm. from caudal extremity; longitudinal diameter of sucker 0,186 mm. Caudal expansions quite rudimentary. Twentytwo papillae arranged in two paralely rows, beginning at the level of the middle part of the rim of sucker; three preanal, ventral; a very large one at the level of the middle of the rim of sucker two smaller ones near the anus: two voluminous ones ad-anal, lateral, six post-anal; of which four ventral and two lateral; of the four ventral ones two large ones near the anus and two smaller ones close to the caudal extremity; of the lateral, one voluminous at the height of the third ventral papilla counting from the anus, one very small one between the two last ventral. Spicules equal, in shape long and wide, almost rectilinear, with very sharp-pointed and slightly curved distal extremity; length 0,558 mm. width 0,038 mm Gubernaculum curvilinear, measuring 0,228 mm. Anus 0,232 mm. from posterior extremfty.

Female: Excretory pore 0,560 mm. from anterior extremity. Tail rectilinear, aculeiform, followed by chitinous appendix, 0,186 mm. long. Vulva small, slightly prominent, opening beyond the middle part of body. Ovijector slightly developed, with muscular layer. Vestibulum retort-shaped. Horn winding difficult to see on account of the hooklets which almost cover it and hide it from view. Uterus duplex, with divergent branches, coiling a great many times; the coils are full of eggs in different degrees of segmentation and reach from the nerve collar to beyond the anal orifice. Almost round, thin-shelled eggs containing embryos before they are laid; length 0,068 mm. width 0,060 mm. Anus 1,176 mm. from posterior extremity.

Development: unknown.

Habitat: intestine of *Trogon viridis* L.

Comes from Rio de Janeiro (Angra dos Reis).

Material: Helminthological collection of the Instituto Oswaldo Cruz nº. 1076.

36. Subulura bentocruzi.

(Plate XVIII, figs. 1, 2 and 3).

Dimensions:

♂ Length: 7,7 mm.—16 mm.; width: 0,436 mm. in middle part of body.

♀ Length: 13 mm—21 mm.; width: 0,643 mm. at the height of vulva.

Body yellowish-white, with the cephalic part a little curved towards dorsal surface; caudal part almost straight in the female, ventrally curved in the male. Cuticle very thin, with transversal striations, 0,0029 mm. distant one from the other. Lateral cephalic expansions slightly developed, reaching to a little beyond the oesophagean bulb, about 1,970 mm. long. Nerve collar 0,372 mm. from anterior extremity. Excretory pore a little behind the nerve collar. Mouth hexagonal, with indistinct lips, surrounded by six papillae, small, equal in size and arranged in two

lateral rows. Buccal capsule small, cylindrical, the walls covered with strong chitinous plates. Three blunt, subquadrangular teeth, 0,020 mm. long, are found at the base of the capsule. Oesophagus rectilinear, club-shaped at posterior extremity; the walls not very thick in the anterior part, become more muscular in the posterior part; oesophagean canal spacious and weakly chitinised in nearly all its length; the chitinous layer becoming thicker in the last third; length: 1,495 mm. Bulb spheroid, with medium diameter of 0,190 mm.; with three subtriangular, chitinous plates. Initial segment of intestine dilated, directly connected with the bulb in the shapede of a winding tube, the terminal part being differentiated in a small rectum with light walls.

Male: Excretory pore opens 0,604 mm. from cephalic extremity. Depth of buccal capsule 0,038 mm. Posterior extremity strongly curved towards ventral surface, ending in a rectilinear appendix, measuring 0.093 mm. Sucker elliptical, without chitinous formation, its antero-posterior diameter equal to 0,280 mm. the lower limit is 0,883 mm. from caudal extremity. Caudal expansions very much atrophied. Eleven pairs of papillae thus arranged: three voluminous preanal ones: one at the level of the higher third of sucker, one equally distant from the anus and the lower rim of sucker, one at the cloacal orifice; two large ad-anal ones, one lateral, the other ventral; six post-anal: four ventral, of which three are larger than the others and equidistant, the lesser one being near the posterior extremity; two lateral ones, a large one at the height of the third ventral papillae another small one between the two last post-anal ones. Spicules two, unequal, with tapering point, the lesser one behind, about 4,5 of the greater one; length: larger one 1,524 mm.; lesser one 1,054 mm. Accessory piece, slightly ventrally curved, the outer extremity is sharp-pointed and the inner one more dilated; length: 0,169 mm. Anus 0,254 from caudal extremity.

Female: Excretory pore 0,651 mm. from anterior extremity tail sharp-pointed slightly bent towards ventral surface followed by appendix 9,140 mm. Vulva opening above the middle of body, prominent. Ovijector comparatively large (0,899 mm.) from the vulva, opening towards the cephalic extremity. Vestibulum ampulliform with the neck turned at right angle; lumen covered with a strong layer of chitin 0,334 mm. Sphincter small. Horn 0,541 mm. long, with very muscular thick walls, which gradually diminish in thickness, until they are as thin as the uterine walls with which they continue. Uterus duplex, with diverging branches and hooklets, reaching to the first part of intestine and traversing the general cavity till beyond the anal orifice. Eggs ellipsoid, very thin-shelled, containing embryos when emitted; length: 0,083 mm width: 0,049 mm. Anus 0,156 mm. from posterior extremity.

Life History: unknown.

Habitat: intestine of *Trogon sp.* and *Trogon variegatus* Spix.

Came from: S. Paulo (Baurú). Ceará (Museu Rocha).

Material: Helminthological Collection of the Instituto Oswaldo Cruz nos. 1078 and 1079.

37. Subulura carlosi Barreto 1918.
(Plate XIX figs. 1, 2 3 and 4).

Dimensions:

♂ Length: 9 to 10 mm. width: 0,29 mm.

♀ Length: 10 to 15 mm. width: 0,35 mm.

Body yellowish white, filiform, anterior part almost rectilinear or slightly bent towards dorsal surface, posterior part straight in the female, strongly curved in the male, ending in a sharp-pointed appendix. Cuticle fine, delicately transversally striated, striations 0,0021 mm. distant from each other. In both sexes are found lateral cephalic small expansions, dwindling away at the level of the posterior extremity of oesophagus, their length is 0,93 mm. and width 0,051. Nerve collar at anterior third of oesophagus. Excretory pore opening on ventral surface, very near the nerve collar, between the latter and the posterior extremity of oesophagus. Mouth with small lips not clearly seen and six small

papillae two larger and four smaller ones. Continuing the buccal orifice, a short vestibulum the walls of which are covered with thick chitinous cylindrical plates; it is more dilated in the posterior part where may be found three small subquadrangular, slightly sharp, teeth. Oesophagus rectangular with piriform dilatation at posterior extremity; oesophagean walls muscular, especially at the dilatation: lumen of oesophagus spacious and little chitinized in the two anterior parts, narrowing and covered with a thick layer of chitin in the posterior third of oesophagus 1,10 mm. Bulb sub-spherical, lodged in the anterior extremity of intestine, which is dilated to receive, it; it communicates with the oesophagus by means of a short passage with transparent lumen; three triangular strongly chitinised plates can be distinguished in the interior of bulb. Intestine dilated in the initial part, rectilinear, ends in a small rectum with thin walls.

Male: Caudal extremity funnel-shaped, curved, like a hook, towards the ventral surface. Sucker small with chitinous rim, 0,212 mm. long; inferior limit 0,465 mm. from posterior extremity. Caudal expansions very reduced. Genital papillae eleven pairs in number arranged thus: three large ones pre-anal, ventrally of which one is at the level, of the middle part of the border of the sucker, one half-way between sucker and anus, another just above the anus: two adanal, voluminous ones: one ventral, the other lateral: six post-anal: four ventral and two lateral; the ventral ones are arranged in two groups, each group has two papillae: one group of larger papillae, near the cloaca another with smaller papillae near the caudal extremity; the lateral ones occupy the following positions: a large one between the two groups of ventral papillae, a very small one between the third and fourth ventral ones. Spicules two, very *unequal* of different length, width, and shape, accompanied by accessory piece; the larger spicule is sinuous, thin, weakly chitinised and spirally rolled, evaginated, about three times as long as the other; this latter one is very strongly chitinised, not curved, and like the larger one has the proximal extremity dilated, and the distal one sharp-pointed; length of larger one: 2,540 mm.; width: 0,016 mm; of lesser one: length: 0,960 mm.; width: 0,025 mm. accessory piece 0,169 mm. long. Anus opens at the side of a little ridge, (0,186 mm.) from caudal extremity.

Female: Posterior extremity rectilinear, aculeiform. Vulva not prominent, near middle of body, more or less at the junction of the anterior 2/4 with posterior 3/5. Ovijector, 1,136 mm. long, bent from anterior towards posterior extremity. The vestibulum, internally covered with a thick layer of chitin, is formed by two very characteristic pieces. (vid. Plate XIX, fig. 2) and is in all 0,254 mm. in size. Sphincter short, nearly always containing an egg, length 0,135 mm. Horn very muscular, curving before opening into uterus. Uterus dichotomous, with diverging branches; uterine hooklets reach from first part of intestine to a little above anus. Eggs containing embryos when emitted, elliptical, with very thin shells 0,084 mm. long and 0,067 mm. wide. Anus 1,156 mm. distant from caudal extremity.

Development: unknown.

Habitat: intestine of *Piaya cayanna* (L). (1)

Provenience: State of Rio de Janeiro, (Angra dos Reis, Ilha Grande)

Material: from Helminthological Collection of the Instituto Oswaldo Cruz, nos. 1094 to 1098.

38. Subulura olympioi Barreto, 1918.

(Plate XX, figs. 1 to 5).

Dimensions:

♂ Length: 5,0 to 8,4 mm.; width: 0.411 mm.

Note. 1. The nematode parasite of *Piaya cayana* (L) *(Cuculus Tringuaza* ought to bear the name *Subulura forcipata* (Rud.). has according to this author equal spicule and lateral expansions. In our material, on the contrary the male has spicules widely different in length and aspect. We therefore think that it is not to be identified with *Ascaris forcipata* Rud. *(Specimina in intestinis caculorus seniculi, naevii et tringuaza reperta. . . . .)* judging it to be a new species of *Subulura.*

♀ Length: 7,7 to 15,6 mm.; width: 0,462 mm. at height of vulva.

Body rectilinear, filiform, yellowish-white, tapering at the extremities. In the greater part of specimens the cephalic end is cylindro-conical, curved towards the dorsal surface; the caudal end sharp-pointed and ending in a chitinous appendix, straight in the female; ventrally curved like a hook in the male. Cuticle of medium thickness (0,009 mm.), the superficial layer transversally striated; striations being 0,003 mm. distant from one another. Lateral cephalic expansions disappearing a little below the bulb. Nerve collar situated approximately at the height of the junction of the anterior 1/4 and the posterior 3/4 of oesophagus. Excretory pore opens on ventral surface, beyond the nerve collar, at the limit of the anterior 1/3 of oesophagus. Mouth without clearly individualised lips, surrounded by six small papillae, arranged in two lateral rows. Buccal capsule narrow, the posterior segment being larger; walls clothed with thick chitinous plates; teeth three in number, club-shaped, found at the entrance to the oesophagus. Oesophagus rectilinear, musculature very accentuated, dilated like a pear ar posterior extremity. Bulb spherical depressed towards posterior direction, communicating with oesophagus. Intestine: anterior end chalice-shaped, containing the bulb and sloping gradually towards the ventral surface, where it ends in a short rectum.

Male: Buccal capsule 0,041 mm. long: 0,029 wide. Nerve collar 0,296 mm. from cephalic extremity. Excretory pore 0,465 mm. from same. Oesophagus 0,899 mm. long. Antero-posterior diameter of bulb 0,179 mm. Transversal diameter: 0,231 mm. Caudal expansions slightly developed. Sucker small, elliptical, without chitinous border, 0,034 mm. long. and 0,033 wide. The posterior limit 0,465 mm. from caudal extremity. Eleven pairs of papillae thus arranged, three large, equal pre-anal ventral: one at the side of sucker at the height of its higher boundary, one just below the sucker, another a litle above the anus; two ad-anal large equal ones; one lateral, the other sub-ventral; six post-anal five ventral and one lateral of the ventral ones: two large ones at the junction of the anterior 1/3 and the posterior 2/3 of the post-anal segment of tail, one at the level of the junction of the anterior 2/3 and the posterior 2/3 of the same, two small ones at posterior extremity, one very small one below the second post-anal ventral, at the point of insertion of the large lateral papillae. Spicules two, *equal*, strongly chitinised, transversally striated with sharp-pointed distal extremities; generally evaginated; length, 0,899 mm. width: 0,016 mm. Gubernaculum straight, short (0,127 mm). Anus 0,228 mm. from posterior extremity.

Female: Buccal capsule 0,067 mm. long. 0,033 mm. wide. Nerve collar 0,304 mm. from cephalic extremity. Excretory pore 0,474 mm. from same. Oesophagus 1,128 mm. large. Bulb: longitudinal diameter 0,084 mm.; transversal diameter, 0,093 mm. Vulva not prominent, small, opening a little above the middle of body. Ovijector short, (0,693 mm.), directed from the vulval orifice towards the anterior extremity, vestibulum muscular with spacious lumen, 0,211 mm. long; sphincter small (0,127 mm.); Horn short, abruptly bent towards the tail before reaching the uterus, 0,338 mm. in size. Uterus double, numerous coils enveloping intestine, filling the general cavity from bulb till beyond the anus. Ovaries arranged in opposite directions, in front of vulva. Eggs elliptical, thin-shelled, embryo developed before laid; length 0,067 mm.; width: 0,050 mm. Anus 0,976 mm. from posterior extremity.

Life History: unknown.

Habitat: Intestine of:

Crypturus parvirostris Wagler.
Rhynchotus rufescens (Temm).
Nothura maculosa (Temm).

Source: Minas Geraes (Lagôas Periodicas de Nova Granja: Lassance).

Material: Helminthological Collection of the Instituto Oswaldo Cruz, nos. 1109 to 1118.

39. Subulura plotina Baylis, 1919.

Dimensions:

♂ Length: 8,2; width: 0,34 mm.

♀ Length: 14,2; width: 0,45 mm.

Body cylindroid, tapering at extremities. Cephalic expansions lanceolate in both sexes, going from cephalic extremity to bening of oesophagean bulb. Bulb hexagonal. Buccal capsule small, containing three very small teeth at entrance of oesophagus. Oesophagus with oval bulb, with distinct anterior part and posterior part fitting into first part of intestine. Mouth without lips, surrounded by six (?) small papillae.

Male: Excretory pore 0,45 mm. from anterior extremity. Oesophagus together with bulb, 1,25 mm. long. Tail without alae, 0,2 mm, long. Sucker long without horny rim, 0,4 mm. from anus. Spicules equal 0,9 mm. long, followed by accessory piece (two accessory pieces ?) of which the largest part measures 0,15 mm. Ten pairs of caudal papillae: six post-anal, one ad-anal and three pre-anal; of these, the third pre-anal (counting from caudal extremity) and the ad-anal are lateral. The foremost of the pre-anal near the sucker, a little in front of its middle part.

Female: Excretory pore 0,65 mm. from head. Oesophagus 1,5 mm. long. Tail 0,8 long mm. Vulva in the middle third of body 5,8 mm. from head. Eggs 65×52.5 micra to 75×55 micra. Uteri distended to beyond anus.

Host: Plotus rufus (Darter).

Origin: Uganda (Africa).

GENUS OXYNEMA Linstow, 1899.

Synonims.

Heterakis pr. part. SONSINO 18889 – p 227.

Oxynema LINSTOW 1699 – p. 19.

Subulura (Oxynema) RAILLIET & HENRY 1913—p. 681.

Subulura pr. parte. TRAVASSOS 1913 – p- 19.

Oxynema TRAVASSOS 1914 – p. 138 & 139.

Allodapa pr. parte. SEURAT, 1915 – p. 126.

Subulura pr. parte. HALL, 1916 – p. 52, 54 and 60.

Oxynema BARRETO 1917 b. p. 305.

Historical Notes.

Oxynema was the name given by LINSTOW in 1899 to a new genus of worms, the type species of which was represented by a nematode from the Museum of Berlin: *Oxynema rectum* n. sp. collected by HEMPRICH and EHRENBERG in two Egyptian canine animals: *Vulpes (Megalotes) zerda* Zimm. and *Vulpes (V) vulpes* var. *aegpptiaca* (Desm). Placing this new genus in the group of meromyarians of SCHNEIDER, LINSTOW supposes them to be related to the *Oxyuridae.*

Comparing the characteristics attributed to *Oxynema rectum* with the description of *Heterakis crassispiculum* SONSINO (1889), RAILLIET & HENRY (1913) observed a great likeness between the two worms which differ only slightly in size, in the number of papillae and in the number of their eggs.

These authors point out that LINSTOW considered his *Oxynema* as a meromyarian type on acount of the external aspect of the musculature and did not seem to have studied the arrangement of the muscular elements in transvetsal cuts.

RAILLIET & HENRY speak of *Heterakis boueti*, found by GENDRE in the intestine of an African squirrel *(Xerus) erytropus* Geoff.), which presentis the greatest affinity with the parasites of the canine animals (Presence of right spiculum followed by gubernaculum) and is consequently doubtlessly allied to them.

Taking the great number of characteristics, common to the genera *Oxynema* and *Subulura*, into consideration, RAILLIET & HENRY propose that the first be considered as a simple subgenus of the latter, that is as a *Subulura* which had almost or completely, lost one of its spicules.

TRAVASSOS (1914) is of a different opinion, considering that *Oxynema* ought to be considered as a separate genus, sub-ordinate to the sub-family *Subulurinae.*

SEURAT, [1915] adopts the opinion of RAILLIET & HENRY and thinks that Oxynema is not even worth considering a new Sub-genus. This author includes the species *crassispiculum (Oxynema rectum)* in the genus. *Allodapa* and does altogether away with LINSTOW'S genus.

HALL (1916) agrees with SEURAT. Sharing the opinion of TRAVASSOS, we do not agree with those who seek to do away with the genus *Oxynema*. The descriptions by SONSINO and LINSTOW are altogether insufficient. GENDRE'S minutious memoranda (1911) about the copulatory organs of the male of *Heterakis boueti*, accompanied by illustrations of the various parts described, cannot be stigmatised as wanting in precision. Whá therefore, should we not admit amongst *Subuluriniae* the presence of some representants possessing one spicule instead of the two male copulatory organs, since we know that other nematodes (Oxyurians for ex.) have only one spicule?

We, therefore, reestablish the genus *Oxynema* as part of the Sub-family *Subulurinae*.

Diagnosis of the Genus.

Subulurinae: Mouth circular with rudimentary lips, and six cephalic papillae. Buccal capsule, cylindrical, ample, contains three teeth in the lower part. Oesophagus accompanied by bulb.

Male: One spicule followed by gubernaculum. Sucker without chitinous rim. Caudal expansions slightly developed. Genital papillae vary in number from 10 to 11 pairs. *Female:* Vulva prominent, situated in middle part of body. Eggs almost round, embryos developed when emitted.

Habitat: Intestine and caecum of Mammals (Carnivora, Rodentia.)

Type-species: *Oxynema crassipiculum* (Sonsino, 1889).

Catalogue of species.

1. Oxynema crassispiculum (Sonsino, 1889) Barreto, 1917.

Heterakis crassispiculum, Sonsino, 1889 p. 227.

Oxynema rectum, Linstow, 1899-p. 19, Pl. 5, fig. 56.

Subulura (Oxynema) recta, Railliet & Henry, 1913-p. 681.

Subulura (Oxynema) crassispiculum, Railliet & Henry, 1913-p. 682.

Allodapa crassispiculum, Seurat, 1915-p. 126.

Allodapa crassispiculum, Hall, 1916-pp. 52 & 60.

Oxynema crassispiculum, Barreto, 1917-1917 b.-p. 305.

Habitat: caecum of *Vulpes (Megalotis) zerda* Zimm. and *Vulpes (Vulpes) vulpes* var. *aegyptiaca* Desm.

Geographical distribution: Africa (Egypt).

2. Oxynema boueti (Gendre, 1911) Barreto, 1917.

Heterakis boueti Gendre, 1911-p. 74, text. figs. 1 to 6 p. 75.

Subulura (Oxynema) boueti, Railliet & Henry, 1913-p. 382.

Subulura boueti, Travassos, 1913-1913 a. p. 19-1913 b. p. 298.

Subulura boueti, Hall, 1916.-p. 54. text-figs. 60 and 63.

Oxynema boueti, Barreto, 1917-1917 b. p. 305.

Habitat: Intestine of *Xerus (X.) erythropus* Geoff.

Geographical distribution: Africa (Dahomey – Agonagon).

Description of the species.

Oxynema crassispiculum (Sonsino, 1889) Barreto, 1917.

(Pl. XXX, fig. 7.)
XXI.

Dimensions:

♂ Length: 12 mm. (Sonsino). 5,94 mm. (Linstow). Width: 0,35 mm. (Sonsino), 0,21 mm. (Linstow).

♀ Length: 18 mm. (Sonsino), 12,21 mm. (Linstow). width: 0,35 mm. (Sonsino), 0,28 (Linstow).

Body subcylindrical, filiform, tapering at extremities: anterior extremity obtuse; posterior sharp-pointed especially in the female,

curved in a semi-circle in the male. Mouth round, surrounded by six small papillae. Buccal capsule long. Oesophagus ending in spherical bulb, communicating with the intestine, the initial part of which is dilated by means of a narrow canal. Excretory pore 0,55 mm. from buccal orifice.

Male: Tail bent towards the ventral surface; 1/50 of the length of body. Oesophagus 1,5 of body (LINSTOW) measuring 1,20 mm. (SONSINO). One spicule strongly chitinised, curved, transversally, striated, ending in a triangular point, 0,50 mm. (SONSINO) to 0,56 mm. (LINSTOW) long. Accessory piece corresponding to 1,3 of spicule (SONSINO); 0,15 mm. long (LINSTOW). Sucker elliptical, without chitinous rim. Ten pairs of genital papillae two of which are pre-anal.

Female: Tail equal to 1/15 of body. Oesophagus 1,5 mm. (SONSINO), or 1,9 (LINSTOW), long. Nerve collar 1,20 mm. from caudal apex (SONSINO). Vulva a little in front of the middle of body dividing it in the proportions 11:14. Ovijector voluminous, directed backward from front, composed of annular muscular fibres. Uterine hooklets reaching from oesophagean bulb to a short distance from anus. Eggs elliptical hatched; 0,045 mm. (SONSINO) to 0,065 mm. (LINSTOW) long, and 0,030 mm. to 0,047 mm. wide.

Habitat: Caecum of *Vulpes (Megalotis) zerda* Zimm. and *Vulpes (V) vulpes* var. *aegyptiaca* Desm.

Geographical distribution: Egypt.

Oxynema boueti (Gendre 1911) Barreto, 1917.

(Pl. XXI, figs. 1 to 6).

Dimensions:

♂ Length: 9,55 to 11,10 mm.; width: 0,44 to 0,48 mm.

♀ Length: 15,52 to 18,78 mm.; width: 0,60 to 0,64 mm.

Body straw-coloured, slender at extremities in both sexes, especially at posterior one. Cuticle finely striated. Lateral expansions very short. Cephalic extremity truncate, continuing the body. Mouth wide, circular, without lips, surrounded by six fungiform, almost equidistant papillae: four submedian and two lateral. Buccal capsule spacious, cylindrical, with three strong chitinous teeth. Pharynx short. Oesophagus thickening gradually until the posterior part; slightly dilated. Bulb spherical. Intestine has initial dilatation larger than bulb, running straight to anus. Excretory pore on ventral surface on the anterior third of oesophagus.

Male: Oesophagus equal to 1/55 of total length. Tail equal to 1,23 to 1,31,7. Posterior region of male ventrally curved, ending in sharp-pointed conical tail, followed by appendix. Sucker oval, without chitinous rim. Caudal bursa formed by two highly developed expansions, reaching from sucker to terminal appendix. Eleven pairs of genital papillae, five pre-anal and six post-anal. Pre-anal: three sub-median, one at the level of the middle part of sucker, another at equal distances from sucker and anus, the last near the cloaca, two lateral near the side of the anus. Post-anal: two large sub-median in anterior part of tail, two smaller ones near caudal extremity, two intermediate lateral ones near the pre-anal, the fourth post-anal is the smallest of all. One spicule only, bow-like, strong and delicately transversally striated, measuring 0,94 mm. formed by two distinct parts one shorter cylindrical, ending in a funnel-shaped dilatation; the other sharp-pointed furnished with lateral expansions. Accessory piece hollowed out, abruptly dilated at base.

Female: Oesophagus 1/7,6 to 1/8,4 of body; Tail straight, conical, ending in appendix like that of the male, corresponds to 1/1,9 1/13,8 of total length. Vulva very prominent, visible to the naked eye, transversal, opening in the anterior half of body; about 1/2,5 of body. Uterus double. Eggs round, thin-shelled, embryos developed when laid, 0,062 long and 0,052 wide.

Habitat: Intestine of *Xerus (X) erythropus* Geoff.

Geographical distribution: Africa: Dahomey, Agouagon.

Genus Cissophyllus Railliet & Henry, 1912.

Synonyms.

Cissophyllus, Railliet & Henry, 1912. p. 253.
Cissophyllus, Railliet & Henry, 1813. p. 679.
Cissophyllus cissophyllus Travassos, 1913—1913 [a]. pp. 5, 6, 24 and 25—1913, [b]. pp. 271, 272, 309 and 310.
Cissophyllus, Travassos, 1914,—p. 138.

Diagnosis.

Mouth furnished with three complex lips, the dorsal one trident-shaped, the lateral ones furnished with plates. Oesophagus divided in to three distinct parts, the last one ending in bulb.

Male: Without caudal expansions. Spicules almost equal, accompanied by accessory piece. Pre-anal sucker drawn out lengthways, without horny rim.

Female: Vulva situated in the posterior third of body. Uteri parallel, bent towards anterior extremity. Eggs thin-shelled, segmented when laid.

Habitat: intestine of *Chelonians.*

Type-species *Cissophyllus laverani* RAILLIET & HENRY 1912.

Catalogue of species.

1. Cissophyllus laverani RAILLIET & HENRY, 1912.

Cissophyllus laverani Railliet & Henry 1912, p. 253.
Cissophyllus laverani Railliet & Henry, 1912 p. 679.
Cissophyllus laverani Travassos, 1913—1913 [b]. p. 25; 1913 [b]. p. 310.

Habitat: Testudo emys Schleg.

Geographical distribution: Asia (India Malasia).

2. Cissophyllus (?) roseus (Leidy, 1851) Barreto, 1917 (1)

Cucullanus roseus Leidy, 1851—p. 155.
Cucullanus roseus Leidy, 1865—p. 54.
Cucullanus roseus Diesing 1860—p. 713.
Cucullanus raseus Railliet & Henry, 1915—1915 b. p. 451.
Cucullanus roseus Leidy 1886—p. 313.

Habitat; intestine of *Testudo* sp. *Testudo emys* Schleg.

Geographical distribution: Asia (Java).

Cissophyllus laverani Railliet & Henry, 1912.

(Pl. XXII, figs. 1, 2 and 3).

Dimensions:

♂ Length: 25 to 31 mm.; width: 1,5 to 1,6 mm.

♀ Length: 24 to 31 mm.; width: 1,6 to 1,8 mm.

Body cylindrical somewhat attenuated at extremities. Tegument opalescent, but still transparent enough to allow one to see the white colouring of the genital organs and the dark red of the digestive tube. Cuticle delicately transversally striated; the striations being 0,0015 mm. distant from one another in the cephalic region where the cuticle separates from the sub-jacent layer, the distance between the striations reaches 0,0065 mm. Polymiarian musculature. Very complex mouth (vid. figs. 1 and 2), lengthened dorso-ventrally. Dorsal commissure occupied by a strong first tooth, trilobate like an ivy-leaf, moving round the base; the lateral borders re-inforced by chitinous formation, segmented in four departments with a great number of plates arranged in groups and bent towards the centre. Six cephalic papillae: two double

Note (1). Leidy and Railliet & Henry give as hosts two tortoises from Java: *Testudo sp.* and *Manouria fusca* According to the catalogue of the British Museum, the species *Manouria fusca* (Gray) corresponds to *Testudo emys* Schleg., This latter name ought to be adopted in preference to the other. This being the case perhaps *Cissophyllus laverani* of Railliet & Henry might be identified with *Cucullanus roseus* of Leidy, though we cannot affirm it as we are uhable to consult Leidy's descriptions.

lateral ones; four sub-median having on the inside a kind of small pad. Oesophagus measures 3,6 mm. to 3,8 mm., is composed of three more or less equal parts: the two first are distinguished by the degree of chitinisation, the second by the darker colouring, the last by its greater diameter ending in a spherical bulb, which contains chitinous plates. Initial part of intestine dilated, in direct communication with oesophagean bulb, afterwards constricted and with uniform diameter to the anus.

Male: Tail curved ventrally. Ample cloaca with prominent rims, opening 0,650 mm. from caudal extremity. Sucker but slightly developed, longitudinal, cleft-like, situated in the middle ventral line, 2, 3 – 2,5 mm. above the cloaca. Spicules two, equal, 1,675 mm. long and 0,095 – 0,110 mm. accompanied by hollowed out accessory piece, which measures about 0,400 mm. in length and 0,200 in width in the middle part and 0,160 mm. in posterior part. No caudal expansions. Caudal papillae eleven pairs: six pre-anal and five post-anal.

Female: Tail rectilinear, abruptly attenuated from the anus, 0,900 mm. from posterior extremity, with a papilla on each side, corresponding to the dorsal papilla of the caudal group of the male. Vulva opens a little behind the posterior third. The two uterine branches are turned in parallel direction towards the front. Eggs oblong, thin-shelled, segmented in 2,4 or even 8 blastomeres when emitted, measuring 0,10 to 0,117 length and 0,052 to 0,062 mm. in width.

Habitat: Testudo emys Schleg.

Geographical distribution: Asia (India, Malasia).

Cissophyllus roseus (Leidy, 1851) Barreto 1917. (1)

Dimensions:

♂ Length: 21 mm.: width: 1,5 mm.

♀ Length: 31 mm.; width: 26 mm.

Body spindle-shaped, rectilinear, pink-red in colour: anterior apex obtuse, tail straight, conical and pointed.

Habitat: intestine of *Testudo sp. Testudo emys* Schleg.

Geographical distribution: Asia (Java.)

Cissophyllus (?) penita (Leidy, 1889), Barreto 1917.

Dimensions:

♂ Length: 10,50 mm.; width: 0,385 mm.

♀ Length: 10,50 to 14,70, width: 1,05 mm.

Body cylindrical, tapering at extremities, more attenuated in the anterior part. Oesophagus long, cylindrical, followed by spherical or oval bulb; length of oesophagus, 1,75 mm.; width, 0,125 mm. Bulb: 0,25 mm. Initial part of intestine dilated.

Male: tail strongly curved, the end being near the genital opening; has four pairs of papillae. Spicules, extremely large: 0,08 mm.

Female: Tail long, straight, conical, tapering, 1,25 mm. long. Vulva in the posterior third of body.

Habitat: intestine of *Chrysemys scripta* Schoeff.

Geographical disiribution: United States of North America.

Genus HETEROXYNEMA Hall, 1916.

Synonyms.

Heteroxynema Hall, 1916 – p. 56 to 60 text-figs. 64 to 69.

Diagnosis of the genus.

Subulurinae: Mouth with three lips, three rudimentary teeth, not always present. Cervi-

Note (1).—We translate Diesing's diagnosis, although very incomplete; unfortunately, we could not obtain Leidy's work.

Note 2.—Leidy and Railliet & Henry give as hosts, two tortoises from Java: *Testudo* sp. and *Manouria fusca*. According to the Catalogue of Chelonians published by the British Museum, the species *Manouria fusca* of Gray corresponds to *Testudo emys*. Schleg. Perhaps then. *Cissophyllus laevrani* of Railliet & Henry ought to be identified with the species *Cucullanus roseus*, though we cannot assert this as we were unable to consult the descriptions by Leidy.

cal expansions present. No spicules nor gubernaculum. Pre-anal fusiform sucker without chitinous rim, but a border of delicate cuticular membrane at the anterior and posterior limits, as if the sucker had two cuticular membranes. Vulva at the junction of the anterior and middle thirds of body.

Habitat: intestine and caecum of Mammals (Rodents).

Type-species one: Only known and *Heteroxynema cucullatum* Hall, 1916.

Habitat: large intestine and caecum of *Eutamias amoenus operarius.*

Geographical distribuition: Sant'Acacio, Costilla Country Colorado.

Heteroxynema cucullatum Hall, 1916.

Dimensions:

♂ Length: 2.786 to 4,47 mm.; width: 0,49 t, 0,170.

♀ Length: 7,36 to 7,9 mm.; width: 0,345 to 0,415.

Cuticle transversally striated. Lips three, simple, one dorsal and two latero-ventral ones, each with a medium papilla, apparently double and separated by a papilla from the adjacent lips. Buccal capsule simple and shallow. Oesophagus club-shaped, thickening gradually towards the posterior extremity and ending in a bulb, which is sometimes deepset in the anterior dilated extremity of intestine. Cervical alae prominent, beginning at the base of lips and winding towards the ventral surface.

Male: Diameter of head about 0,065 mm. Oesophagus without bulb, 0,355 0,515 mm. long and 0,050 to 0,070 mm. wide. Bulb 0,136 mm. to 0,162 mm. long and 0,112—0,145 mm thick. Nerve collar 0,130 mm. from cephalic extremity. Caudal expansions weakly developed. The greater part of the genital papillae are grouped round the cloaca; one pair isolated, 0,085—0,112 mm. from posterior extremity. Genital opening 0,235 mm. from extremity. Six ad-anal papillae: two larger external ones and two lesser exactly median ones at each side of these. Just behind these papillae is a sub-median pair followed by a single papilla on the middle line. No pre-anal papillae were observed, the total number being eleven. No spicules. Sucker fusiform, having on the lateral margins two cuticular, fenestrated membranes apparently interrupted in the middle line at the anterior and posterior extremities; length of sucker: 0,112—0,130 mm.; width: varies on acount of the contraction and distension of the flexible borders, the posterior limit is 0,515—0,600 mm. from tail end.

Female: Posterior extremity sharp-pointed. Oesophagus without bulb, 0,430—0,535 mm. long, and 0,056—0,070 mm. thick. Bulb 0,198—0,240 mm. long, 0,130—0,185 mm. wide. Nerve collar 0,170 mm. from cephalic extremity. Anus 1,65 mm. from caudal extremity. Vulva at the junction of the anterior and middle thirds of body, 1,80—3,265 mm. from head. Ovijector with spirally curved walls, initial part turned from the vulva towards the cephalic extremity followed by a tail. Uterus apparently double; uterine hooklets occupy the posterior part of body. Eggs long, with depression on one of the sides, obtuse extremity; 0,098—0,100 mm. long and 0,035 mm. wide.

Habitat: great intestine and caecum of *Eutamias amoenus operarius.*

Geographical distribution: Sant Acacio, Costilla County, Colorado.

Material-type, Number 16637 in Bureau of Animal Industry, U. S. A. Helminthological Collection.

Genus NUMIDICA Barreto, 1918.

Synonymy.

Allodapa SEURAT, 1915 pr. parte.—p. 122 text-figs. 1 to 4.

Historical notice

In the "Comptes rendus des Séances de la Société de Biologie", in 1915, SEURAT describes a nematode living in the caecum of the Algerian fox *(Vulpes* (V) var. *atlantica* WAGNER), under the name of *Allodapa numidica.*

In a note published in "Brazil Medico" (Vol. XXXI—N. 29—p. 243 1917,) we showed that *Allodapa* should be considered as a synonym of *Subulura,* the species included by SEURAT being considered as belonging to this genus. The worm called *Allodapa numidica,* described by this author cannot however be considered as a *Subulura,* as it differs from this group by the following characteristics: the structure of the sucker at the caudal extremity of the male, structure similar to that described by HALL as being also found in *Heterakis oxynema cucullatum,* found in the caecum of *Eutamias amaenus operarius.* The presence of two large cephalic expansions, and of a mouth bounded by three distinctly individualised lips in HALL'S species, forms a great contrast to the absence of lateral, and a buccal cavity with two indistinct lips in *Allodapa numidica* of SEURAT, and prevents the comprising of the worms in the same group. The form and aspect of the long eggs (90—100 micra by 35 micra) of *Heteroxynema cucullatum,* and the round eggs (63 by 58 micra) of the species *numidica* strengthen our opinion.

Diagnosis of the genus.

Subuluriuae: Mouth limited laterally by two indistinct lips, each provided with three papillae. Buccal capsule with thick chitinous walls, divided into two parts: one higher, one lower containing three slightly prominent teeth at the entrance to oesophagus. Oesophagus followed by bulb. No lateral expansions.

Male: Spicules two, unequal, the right-hand one larger, very much chitinised: the left-hand one hardly visible on account of the slight chitinised layr. Gubernaculum narrow strongly chitinised. Sucker represented by an elliptical area transversally striated, surrounded by a rim, covered with small trabecules of cuticle. Genital papillae: 10 pairs.

Female: Vulva prominent, taking up the middle third of body. Ovijector with thick muscular walls, turned towards anterior xetremity. Uterus double, divergent. Eggs round thin-shelled, hatched out.

Habitat: intestine of Mammals (Carnivora).

Type-species and only known one: *Numidica numidica* (Seurat 1915) Barreto 1917.

Geographical distribution: Africa (Algeria Blida).

Numidica numidica (Seurat, 1915) Barreto, 1917. (1)
(Pl. V, figs. 1 to 4),

Synonyms.

Allodapa numidica, SEURAT, p. 1915—p. 122 text-figs. 1 to 4.

Dimensions:

♂ Length: 13,5 mm.; width: 0,370 mm.

♀ Length: 25 mm.: width: 0,375 mm.

Body thin, rectilinear, posterior extremity curved. In the male cuticle, thick delicately transversally striated striations 0,004 mm. from one another. No cephalic lateral expansions. Lateral expansions very distinct, their colour being intense. Excretory pore opening on the middle ventral line, a little beyond the nerve collar. Mouth limited laterally by two indistinct lips, each with three papillae. Buccal cavity with strongly chitinised walls, divided into two parts: one higher, and one lower containing three not very prominent teeth at the entrance of oesophagus. Oesophagus short, slightly dilated like a club in the posterior region, communicating by very short a constriction with the bulb that contains chitinous plates. Nerve collar at the anterior fifth of oesophagus.

Male: Oesophagus (including bulb) equal to 1/9 of body. Tail curved at extremity, sometimes coiled, ending in thin point. Cloaca bounded by two lips, the posterior one voluminous and very prominent, opens at 0,275 mm. from caudal extremity. Caudal expansions short and narrow, limited at sub-terminal region. Sucker represented by an elliptical area, transversally striated and surrounded by a rim of small trabecules of cu-

Note (1) In present paper.

ticle; this formation is surrounded by radiating muscles and is 0,6 mm. in front of cloaca. Pedunculated genital papillae ten pairs in number: four pre-anal and six post-anal; the third and fourth pairs on the outisde, the tenth by the side of sucker. Clearly apparent caudal glands opening just behind the second and in front of the third pair of papillae. Spicules two, unequal the right-hand one strongly chitinised, clearly seen, longish (0,600 mm.) dilated in the free part: the left-hand smaller, (0,440 mm.) weakly chitinised, hardly visible. Gubernaculum spoon-shaped, narrow; greatest width: 0,038 with strong chitinised layer, clearly seen turned transversally 0,180 mm. long.

Female: Buccal cavity 0,060 mm. in depth. Excretory pore 0,610 mm. from cephalic extremity. Oesophagus equal to 1/12,5 of body, 1,8 mm. in size. Bulb 0,250 mm. Tail drawn out ending in an appendix, 0,15 mm. long, i. e. one twelfth of body. Pores of the caudal glands situated in the posterior third of tail. Vulva prominent, opening immediately behind anterior third of body. Ovijector bent forwards, parallel with body, 1 mm. long. Ovijector shows a short vestibulum, remarkable for the thickness of its muscular walls composed of many layers of cells and considerable internal cuticular clothing, continuing the external cuticle of the nematode worm. In the muscular wall of the organ are to be seen four unicelled glands, one ventral and three dorsal, with distinct nucleus and nucleolus and opaque and granulated protoplasma. The sphincter is composed of three parts: the initial part which forms an obtuse angle with the vestibulum and is thickly covered with cuticle, blocking the lumen of the passage so that the eggs passing through come in contact with its walls; the muscular layer is thinner than that of the vestibulum and yet it has three or four layers of cells and two uni-celled glands like those of the vestibulum; the middle region which is characterised by the existence of an organ which gets intensely coloured by picro-carmine and consists of seven or eight long cells, arranged transversally between the muscular tunic which is reduced to two layers of cells) and the internal clothing which is very thin. The terminal region of the sphincter is characterised by the gradual reduction of the external muscular covering. At the passage of the sphincter, the horn is indicated by the presence of high and broad cells with nucleus and nucleoli which are. Very distinct the musculo-epithellial horn consists of a long (10 mm.) narrow tube, which after going forwards for a short distance, bends and curves backwards; it holds a small quantity of eggs arranged all along it; it bi-furcates aud re-unites itself to uteri. Uteri narrow (0,120 – 0,130 mm.) long (the anterior measures 27,5 mm.) (the posterior 30,5 mm.), coiled several times round the intestine: each branch holds about 1,02 eggs, of which those in the distal region have no shell, being wrapped up in the vitelline membrane. The larger of these branches holds 700 eggs arranged in three rows one against the other; the organ narrowing down the eggs 200 in number are arranged in two rows in front about 60 are arranged in one diametrical row; finally the proximal region connected with the horn holds 30 eggs arranged in one plane according to the axis. show the same position which they get in the horn. The distal region of the uteri contain spermatozoids it is not however individualised as a distinct seminal receptacle. Oviducts narrow and short (0,600 mm). Ovaries club-shaped, 4,5 mm. long, divergent, one in front and one behind the vulva, remarkable for their blackish colour, due to the reserve material they contain. Eggs almost round, thin-shelled, smooth, hatched, 0,063 mm. long and 0,058 mm. wide.

Habitat: intestine of *Vulpes (V) vulpes* var *atlantica* WAGNER.

Geographical distribution: Africa (Algeria Blida).

Hosts of the Nematodes of the sub-family SUBULURINAE.

In the following list of the animals hosts of the nematodes of this sub-family, the birds and reptiles bear the names according to

the Catalogue of the Britsh Museum and the Mammals bear the names of the Catalogue of Trouessart: Supplement of 1904. To organize this, we consulted the following works:

BOULENGER, G. A. – Catalogue of the Chelonians, Rhynchocephalians and Crocodiles in the Britsh Museum (Natural History). 1889. – London.

BOULENGER, G. A. – Catalogue of the Snakes in the British Museum (Natural History). 1893 – 1896. – London.

BOULENGER, G. A. – Fishes. In the Cambridge Natural History, Harmer and Shipley. Edits Vol. 7. pag. 588.

BOWDLER SHARPE. – Catalogue of the birds in the British Museum (Natural History). 1879. – London.

BOWDLER SHARPE. – Bulletin of the British Ornithologist's Club. Vol. 9. 1899.

EULER, C. – Descripção de ninhos e ovos das aves do Brasil. Revista do Museu Paulista. Vol. 4. 1900, pag. 9.

GOELDI, E. A. – As aves do Brazil. Rio de Janeiro. 1894.

GOELDI, E. A. – Album de aves amazonicas. Museu Goeldi. Pará. 1894 – 1900.

HAGMANN, G. – As aves brasilicas, mencionadas e descriptas nas obras de SPIX (1824), de WIED (1830 – 1833), BURMEISTER (1854) e PELZELN (1874) na sua nomenclatura actual. Boletim do Museu Goeldi. Vol. 4. 1904 – 1906.

IHERING, H. VON – As aves do Estado de S. Paulo. Revista do Museu Paulista. Vol. 3. 1898, pag. 111.

HERING, H. VON – Aves observadas em Cantagallo e Nova-Friburgo. Revista do Museu Paulista. Vol. 4. 1900. pag. 194.

HERING, H. VON – Catalogo critico-comparativo dos ninhos e ovos das aves do Brasil Revista do Museu Paulista. Vol. 4. 1900, pag. 191.

HERING, H. VON – Novas contribuições para a Ornithologia do Brasil. Revista do Museu Paulista. Vol. 9. 1914, pag. 411.

IHERING, H. VON – Zoologia do Brasil Bibliographia. 1911 - 1913. Revista do Museu Paulista. Vol. 9. 1914, pag. 489. (497).

IHERING, ROD. VON – Fauna do Brasil. S. Paulo. 1917.

PELZELN, A. VON – Zur Ornithologie Brasiliens. Resultate von Johann Natterers Reisen in den Jahren 1817 bis 1835. Wien 1871.

TROUESSART, E. L. – Catalogus mammalium tam viventium quam fossilium. Berolini. 1898 – 1899.

TROUESSART, E. L. – Catalogus mammalium tam viventium quam fossilium. Suplementa ano 1904. Berolini. 1904 – 1905.

Mammalia

I. Ordo PRIMATES.

Cercopithecus (C.) sabaeus (L.)
Cercopithecus (C.) callithrichus (Geoff).
Cercopithecus (Rhinosticus) cephus (L.)
Cercopithecus patas (Schreber.)
Cercopithecus brazzae (Milne Edw.)
Cercocebus (C.) fuliginosus (Geoff.)
Cercocebus (C.) collaris (Gray).
Subulura distans.

Callithrix jacchus (L.)
Callithrix chrysoleucus (Natt.)
Callithrix melanurus (Geoff.)
Midas (M.) bicolor (Spix.)
Callicebus caligata (Natt.)
Subulura jacchi.

II. Ordo PROSIMIAE.

Tarsius tarsius (Erxleb.)
Subulura perarmata.

Galago (G.) galago (Schreber).
Galago (H.) demidoffi (Fischer.)
Galago mossambicus (Peters.)
Subulura otolicni.
Loris gracilis (Geoff.)
Subulura sarasinorum.

III. Ordo CARNIVORA.
Cynictis pennicillata (Cuv.)
Subulura schebeni.
Vulpes (Megalotis) zerda (Zimm).
Vulpes (V). vulpes var. ægyptiaca (Desm).
Oxynema crassispiculum.
Vulpes (V). vulpes var. atlantica Wagner.
Numidica numidica.

IV. Ordo RODENTIA.
Sciurus sp.
Subulura andersoni.
Sciurus prevosti rafflesi (Vigors & Horsfield.)
Subulura pigmentata.
Dipodillus campestris (Levaill.)
Subulura elongata.
Xerus (X.) erythropus (Geoff.)
Oxynema boueti.
Eutamias amœnus operarius.
Heteroxynema cucullatum.

Aves

I. Ordo ACCIPITRES.
1. Sub-ordo STRIGES.
Scops brasilianus (Gm).
Subulura acutissima.
Scops leucotis (Temm).
Subulura similis.
Carine noctua glaux (Sav.)
Subulura noctuæ.
Strix sp.
Subulura lutzi.
2. Sub-ordo FALCONES.
Cariama cristata (L.) (Seriema.)
Subulura allodapa.

II. Ordo PASSERIFORMES.
Sub-ordo PASSERES.
Cyanocorax cayanus (L.)
Subulura papillosa.

III. Ordo PICARIAE.
1. Sub-ordo CORACIAE.
Caprimulgus rufficolis (Temm.)
Caprimulgus europæus (L.)
Subulura subulata.
Caprimulgus rufus (Bodd.)
Caprimulgus nigrescens (Cab.)
Caprimulgus vociferus (Wilson.)
Caprimulgus sp. (Bacuráo).
Podager nacunda (Vieill.) Corução.
Stenopsis candicans Pelz.
Lurocalis semitorquatus (Gm.)
Hydropsalis climacocercus (Tsch.)
Nyctidromus albicollis (Gm.)
Nyctibius ætherus (Wied.)
Nyctibius grandis (Gm.)
Nyctibius jamaicensis (Gm.)
Heliothreptus anomalus (Gould)
Subulura suctoria.
Caprimulgus fossii (Hartl.)
Caprimulgus ægyptius var. saharæ (Erl.)
Macrodipeteryx macrodipterus Afzel.
Subulura leprincei.
Coracias abissinicus Bodd.
Subulura similis.
Eurystomus afer (Gray.)
Subulura recurvata.
Subulura similis.

2. Sub-ordo TROGONES.
Trogon viridis L. (Surucuá.)
Subulura trogoni.
Trogon variegatus (Spix.)
Trogon sp.
Subulura bentocruzi.

3. Sub-ordo SCANSORES.
Malacoptila torquata (Hahn. u. Küst). (João barbudo).
Chelidoptera tenebrosa (Pall.) (Andorinha do matto).
Nonnula rubecula (Spix).
Monacha nigra (Müller).
Monacha morpheus (Hahn. u. Küst.)
Bucco collaris (Lath.)
Bucco macrorhynchus (Gm.)
Bucco tectus (Bodd.)
Bucco tamatia (Gm.)
Bucco striolatus (Pelz.)

Bucco rufiventris (Natt.) (1)
Bucco swainsoni Gray e Mitch (João do matto).
Bucco chacuru Vieill. (João bôbo).
Subulura travassosi.

4. Sub-ordo COCCYGES.
Coccyzus melanocoryphus (Viaill.)
Coccyzus minor (Gm.)
Diploterus nævius (L.)
Guira guira Gm. (Anum branco).
Subulura forcipata.
Piaya cayana (L.) (Alma de gato).
Subulura forcivata.
Subulura carlosi.
Centropus sinensis (Steph).
Subulura rimula.
Centropus monachus (Rüpp.)
Centropus superciliosus.
Subulura similis.
Crotophaga major (Gm.)
Crotophaga ani (L.) (Anum).
Subulura reclinata.

IV. Ordo GALLINAE.
Sub-ordo ALECTOROPODES.
Odonthophorus capueira (Spix) (Capueira, Urú).
Subulura strongylina.
Gallus damesticus (L.
Numida meleagris (L.) (Gallinha d'Angola. Guiné).
Francolinus bicalcaratus (L).
Subulura differens.
Francolinus adspersus (Waterh.)
Subulura poculum.
Francolinus sp.
Subulura gracilis.
Caccabis saxatilis var. chukar (Gray).
Subulura curvata.
Caccabis rufa (L.). (2)
Caccabis petrosa (Gm.). (2)
Subulura seurati.
Numida rikwae (Rshb.) (1)
Subulura acuticauda.

V. Ordo ALECTORIDES.
Houbara undulata (Desf.)
Houbara macqueeni (*nec* Desf.) (Bescht.)
Subulura rima.
Tetrax tetrax (L.)
Subulura halli.

VI. Ordo CRYPTURI.
Crypturus tataupa Temm. (Inhambú chitam).
Crypturus sp. (Inhambú, Inambú)
Tinamus sp. (Macuco).
Subulura strongylina.
Crypturus parvirostris (Wagl.) (Inhambú choróró).
Nothura maculosa (Temm.) (Codorna).
Rhynchotus rufescens (Temm.) (Perdiz).
Subulura olympioi.

Pisces

Ordo TELEOSTEI.
Sub-ordo OSTARIOPHYSI.
Macrones aor (Ham. Buch.)
Subulura macronis.

Reptilia

I. Ordo OPHIDIA.
Xenodon severus (L.)
Subulura annulata.

II. Ordo CHELONIA.
Sub-ordo TECOPHORA.
Testudo emys (Schleg.)
Cissophyllus laverani.
Cissophyllus roseus.
Testudo sp.
Cissophyllus roseus.
Chyrsemys scripta (Schoepff.)
Cissophyllus penita.

(1) According to Natterer apud Stossich 1888. No. Catalogue of Birds of British Museum we do not find this name. According to v. Jhering (vid. Jhering 1914) b. p. 498 perhaps *Bucco swainsoni.*

(2) This name applies to the red partridge of North Africa. Vid. Seurat 19, and 1914 b.

(1) According to Linstow 1901. We did not find this name in the catalogue of the British Museum.

Literature.

BARRETO, A. L. B., 1916. – Nota sobre *Cucullanidæ* nov. fam., Brazil-Medico. Anno 30. 1916. No. 49, p. 388.

BARRETO, A. L. B., 1917 a. – Notas helminthologicas. I. Sobre o genero *Allodapa* Diesing, 1860. Brazil-Medico. Anno 31. 1917. No. 29, p. 243.

BARRETO, A. L. B., 1917 b. Notas helminthologicas. II. Sobre o genero *Oxynema* Linstow, 1899. Brazil-Medico. Anno 31. 1917. No. 36, p. 305.

VAN BENEDEN, 1890 a. – Un Nematode nouveau d'un Galago de la côte de Guinée. Bull. de l'Academy. Ro. Sc. Belgique. Vol. 19. 1890, p. 389. Figs. 1–7.

VAN BENEDEN, 1890 b. – Un Nematode nouveau d'un Galago de la côte de Guinée. Ctrbl. Bakteriol. 1891. Vol. 9, p. 509. Referate.

BAYLIS, H. A., 1919. —Some new Entozoa from birds in Uganda – Ann. Mag. Nat. Hist. vol. 3, 1919. No. 17, p. 457.

BOULENGER, G. A., – Fishes. In the Cambridge Natural History. Harme. and Shipley. Edits. Vol. 7, p. 588.

BOULENGER, G. A., 1889. – Catalogue of the Chelonians, Rhyncocephalians and Crocodiles in the Bristish Museum (Natural History). 1889. London.

BOULENGER, G. A., 1893–1896. – Catalogue of the Snakes in the British Museum (Nat. Hist.). 1893–1896. – London.

BOWDLER SHARPE, 1874. – Catalogue of the birds in the British Museum (Nat. Hist.), 1874. – London.

BOWDLER SHARPE, 1899. – Bull. of the British Ornithologist's Club. Vol. 9. 1899.

COBBOLD, T. S., 1861. – List of the Entozoa, including Pentastomes from animals dying at the Society's Menagerie, between the years 1857–1860 inclusive, with descriptions of several new species. Proc. Zool. Soc. Lond. 1861. p. 117.

COBBOLD, T. S., 1864. – Entozoa: an introduction to the study of Helminthology. – London. 1864.

COBBOLD, T. S., 1876. – Notes on Entozoa. – Part. 4. Proceedings of the Zoological Society of London 1876, p. 294.

CREPLIN, 1853. – Eingeweidewürmer des Dicholophus cristatus. Abhandl. d. Naturf. Gesellch., Halle, 1853. Bd. I, p. 59–68.

DIESING, 1851. – Systema Helminthum. – Vindobonae, 1851, vol., 2.

DIESING, 1860. – Revision der Nematoden. – Sitzungsber. der Math. Nat. der k. Akad, der Wissench. Wien, 1860. Bd. 42. No. 28.

DRASCHE, 1882. – Revision der in der Nematoden-Sammlung des k. k. zoolog. Hofcabinets bef. Original Exem-

plare Diesing's und Molin's. Verhandl der k. k. zool. bot. Gesell., Wien. Jahrg. 1882. Bd. 32, p. 117.

DUJARDIN, 1845. — Histoire naturelle des helminthes ou vers intestinaux. Paris 1845.

EULER, C., 1900. — Descripção de ninhos e ovos das aves do Brazil. Revista do Museu Paulista. Vol. 4. 1900, p. 9.

GEDOELST, L., 1916. — Notes sur la faune parasitaire du Congo Belge — Rev. Zoologique Afric. v. 5. 1916 — fasc. 1 p. 41 text-figs. p. 42, 43.

GEDOELST, L., 1917. — Nematodes parasites du *Sciurus prevosti* de Sumatra — Rev. Zool. Afric. N. 5 — 1917 — fasc. 2. p. 153 text-fig.

GENDRE, 1909. — Notes d'helminthologie africaine, troisième note. Extrait des Procès-verbaux de la Soc. Linn de Bordeaux.—1909.

GENDRE, 1911. — Sur quelques espèces d'Heterakis du Dahomey. Actes de la Soc. Linn. de Bordeaux. — 1911. Tome 65. — Procès-verbaux, pag. 68.

GOELDI, E. A., 1894. — As aves do Brazil. — Rio de Janeiro. 1894.

GOELDI, E. A., 1894—1900. — Album de aves amazonicas. Museu Goeldi. — Pará. 1894 — 1900.

HAGMANN, G., 1904. — As aves brasilicas, mencionadas e descriptas nas obras de Spix (1825), de Wied (1830-1833), Burmeister (1854) e Pelyeln (1874) na sua nomenclatura actual. Boll. Mus. Goeldi. Vol. 4. 1904 — 1906, p. 198.

HALL, M., 1916. — Nematode parasites of Mammals of the orders Rodentia, Lagomorpha and Hyracoidea, Proc. U. S. Nat. Hist. Mus. Vol. 50, p. 1 — 258.

IHERING, H. VON, 1898. — As aves do Estado de S. Paulo. — Revista do Mus. Paulista. Vol. 3. 1898, p. 111.

IHERING, H. VON, 1900 a. — Aves observadas em Cantagallo e Nova-Fiburgo. — Revista do Mus. Paulista. Vol. 4. 1900, p. 149.

IHERING, H. VON, 1900 b. — Catalogo critico-comparativo dos ninhos e ovos das aves do Brasil. — Revista do Mus. Paulista. Vol. 4. 1900, p. 191.

IHERING, H. VON, 1914 a. — Novas contribuições para a Ornithologia do Brasil. — Revista do Mus. Paulista. Vol. 9. 1914, p. 411.

IHERING, H. VON, 1914 b. — Zoologia do Brasil. — Bibliografia 1911 — 1913. — Revista do Museu Paulista. Vol. 9. 1914, p. 489.

IHERING-ROD. VON, 1917. — Fauna do Brasil. — S. Paulo. 1917.

LANE, CL., 1914. — Suckered round-worms from India and Ceylon. — Indian Jour. Med. Res. Vol. 2. 1914, p. 655.

(*) LEIDY, J., 1851. – Descriptions of new species of Entozoa. Proc. Acade. Nat. Sc. Philad. Vol. 5. 1851, p. 155.

(*) LEIDY, J., 1856. – A synopsis of Entozoa and some of their ectocongeners observed by the author. Proc. Acad. Nat. Sc. Philad. Vol. 8. 1856, p. 54.

LEIDY, J., 1885. – On some parasitic worms of birds. Proc. Acad. Nat. Sc. Philad. 1885, p. 9.

LEIDY, J., 1886. – Notices on nematoid worms. Proc. Acad. Nat. Sc. Philad. 1886, p. 308.

LEUCKART, 1858. – Bericht über die Leistungen in der Naturgeschichte der niederen Thiere während des Jahres 1857. – Archiv Naturg. 1858 – Jahrg. 24. Bd. 2, p. 108.

LINNAEUS, 1758. – Systema naturæ etc. – Stockholm, 1758.

LINSTOW, 1883. – Nematoden, Trematoden und Acanthocephalen, gesammelt von Prof. Fedtschenko in Turkestan. – Archiv. f. Naturgesch. 1883. Berlin. Vol. 49. Bd. I, p. 274.

(*) LINSTOW, 1886. – Vermi: viaggio Fedtschenko (lav. russo) Mosca. 1886, p. 15, fig. 24.

LINSTOW, 1899. – Nematoden aus der Berliner Zoologischen Sammlung. – Mitteil. aus der Zool. Mus. f. Naturkunde. – Berlin. 1898 – 1900. Bd. I. Heft. 2.

LINSTOW, 1901. – Helminthen von den Ufern des Nyassa-Sees. Jenaische Zeitschrifft f. Naturgesch. 1901. Bd. 35 – N. F. 28 – Heft. 4, p. 409.

LINSTOW, 1903. – Parasiten, meistens Helminthen, aus Siam. – Archiv. f. Mikroskop. Anat. u. Entwickl. Bd. 62, p. 108.

LINSTOW, 1906. – Nematoden des zoologischen Museums in Königsberg. – Archiv. f. Naturgesch. 1906. – Jahrgang 72. Bd. I, p. 249.

LINSTOW, 1909. – Neue Helminthen aus Deutsch-Südwest Afrika. – Centralblatt f. Bakteriol. etc., Orig., 1909, Bd. 50. Heft. 4, p. 448.

(*) MARCEL, 1857. – Sur des vers intestinaux du Ouistiti. Bull. Soc. Vaud. Sc. Nat. Lausanne. 1956 – 1857. Vol. 5, p. 340 – 341.

(*) MEYER, 1896 a. – New Singhalese nematods. – Jour. Royal Micros. Soc. London. 1896, p. 627.

MEYER, 1896 b. – Neue ceylonische Nematoden aus Säugethieren (Filaria, Strongylus) und Julus (Oxyuris) – Archiv. Naturg. Jahr. 62. 1896, p. 54, taf. 4 – 5.

MEYER, 1896 c. – von Linstow – Bericht über die wiss. Leistungen in der Naturg. Helminthen in Jahre 1896. –

Archiv. Naturg. Jg. 61. 1895. Bd. 2 Hft. 3, p. 107.

MOLIN, 1860. — Trenta specie di Nematodi. — Sitzungsber. d. k. Akad., Wien. 1860. Bd. 40.

(*) PARONA, 1889. — Ann. Museu civico di Genova. Vol. 27. 1889, p. 761.

PELZELN, A. 1871. — Zur Ornithologie Brasiliens. Resultate von Johann. Natterers Reisen in den Jahren 1871 bis 1835. Wien. 1871.

RAILLIET, A., 1895. — Traité de Zoologie Médicale et Agricole. Paris 1895

RAILLIET & HENRY, 1912. — Quelques Nematodes parasites des Reptiles. — Bull. Soc. Pathol. Exot. 1912. Tome 5, p. 251.

RAILLIET & HENRY, 1913. — Essai de Classification des «Heterakidæ». — Extrait du IX Congrès Int. de Zool. de Monaco 1914.

RAILLIET & HENRY, 1915 a. — Sur les Nématodes du genre *Goezia* Zeder. — Bull. Soc. Pathol. Exot. Vol. 8, p. 270.

RAILLIET & HENRY, 1915 b. — Sur les Nématodes du genre *Cacullanus* Raill. & Henry, 1915 *(Cucullanus* Auct., non Muller, 1777). — Bull. Soc. Pathol. Exot. Vol. 8. 1915, p 446.

RATZEL, 1868. — Beschreibung einiger neuen Parasiten. — Archiv. f. Naturgesch. — Berlin 1868. Jg. 34. Bd. I, p. 150.

(*) RUDOLPHI, 1809. — Entozoorum seu vermium intestinalium historia naturalis. — Amstelædami, 1808 — 1810.

RUDOLPHI, 1819. — Entozoorum Synopsis cui accedunt mantissa et indices locupletissimi. — Berolini, 1819.

SCHNEIDER, 1866. — Monographie der Nematoden. — Berlin, 1866.

SCKRJABIN, K. I., — Parasitic Trematodes and nematodes collected by the expedition of Prof. V. Dogiel and I. Sokolow in British East—Africa Petrograd Russia.

SEURAT, 1914 a. — Sur un nouvel habitat et sur la morphologie du *Subulura allodapa* Creplin. — Actes Soc. Biol. 1914. Vol. 77. — Comptos-rendus, p. 154.

SEURAT, 1914 b. — Sur quelques Heterakis d'oiseaux. — Bull. Soc. Hist. Nat. de l'Afrique du Nord, 1914. 6.eme Année. No. 7, p. 195.

SEURAT, 1914 c. Sur deux nouveaux Heterakis du Sud-Algérien. Bull. Soc. Hist. Nat. de l'Afrique du Nord, 1914. 6.eme Année. No. 8, p. 222.

SEURAT, 1915. — Sur deux nouveaux parasites du renard d'Algerie. — Soc. Biol. 1915. Vol. 78. — Comptes-rendus, p. 122.

(*) Papers marked thus, were not read.

SONSINO, 1889. — Studie e Notizie elmintologiche. — Atti Soc. tosc. Sc. nat., 1889. Vol. 6., proc. verb., p. 227.

SONSINO, 1890 a. — Un nuovo Heterakis del Gallus domesticus. — Atti Soc. Tosc. di Sc. nat. — Pisa, 1890. Vol. 7, proc. verb., p. 136.

SONSINO, 1890 b. — Un nuovo Heterakis del Gallus domesticus. — Centralb. Bakteriol. etc. 1891. Orig. Bd. 9. — Referate.

STEWART, 1914. — Studies in Indian Helminthology. No. 1. — Records Ind. Mus. Vol. 10, part. 3, 1914, p. 165.

STOSSICH, 1888. — Il genere Heterakis Dujardin. — Prestapano iz «Glasnika Hrvatskoga Naravos-lovnoga Druztva». 1888.

STOSSICH, 1896. — Il genere *Ascaris* Linné. — Boll. Soc. Adratica Sc. Nat. Trieste. Vol. 17. 1896.

TRAVASSOS, L., 1913 a. — Sobre as especies brasileiras da sub-familia Heterakinæ, Railliet & Henry. — These — Instituto Oswaldo Cruz, 1913. — Rio de Janeiro.

TRAVASSOS, L., 1913 b. — Sobre as especies brasileiras da sub-familia *Heterakinæ* Railliet & Henry. — Memorias do Inst. Oswaldo Cruz. 1913. Tomo 5. Fac. 3., p. 271.

TRAVASSOS, L., 1914. — Novo genero da familia Heterakidæ Railliet & Henry. — Memorias do Instituto Oswaldo Cruz. 1914. Tomo 6. Fasc. 2, p. 137.

TROUESSART, E. L.; 1898 — 1899. — Catalogus Mammalium tam viventium quam fossilium. — Berolini, 1898 — 1899.

TROUESSART, E. L., 1904 — 1905. — Catalogus Mammalium tam viventium quam fossilium. — Berolini, 1904 — 1905. Supplementa anno 1904.

Explanations of Plates. 2–24

Plate 2.

Fig. 1. *Subulura perarmata* RATZEL anterior extremity×75.
" 2. Idem. Gubernaculum×150.
3. Idem. Buccal capsule and teeth 400.
4. Idem. Tail of the male×75.
5. *Subulura distans* SCHNEIDER Head and mouth×90.
6. *Subulura strongylina* SCHNEIDER Tail of male.
" 7. *Subulura similis* GENDRE.
" 8. *Subulura differens* GENDRE.
9. *Subulura distans* SCHNEIDER Tail of male×92.

Plate 3.

Figs. 1. & 3. *Subulura gracilis* LINSTOW
" 2. *Subulura schebeni* LINSTOW.
" 4. *Subulura curvata* LINSTOW.
5. *Subulura poculum* LINSTOW.
6. *Subulura recurvata* LINSTOW.
7. *Subulura acuticauda* LINSTOW
" 8. *Subulura rima* LINSTOW.
" 9. *Subulura rimula* LINSTOW.

Plate 4.

Fig. 1. *Subulura allodapa* DRASCHE. Tail of male×106.
2. *Subulura suctaria* DRASCHE. Tail of male×165.
3. *Subulura acutissima* DRASCHE Tail of male×75.
4. *Subulura allodapa* Male & female. Natural size.
" 5. *Subulnra acutissima.* Cephalic extremity. Buccal capsule.
6. *Subulura papillosa* DRASCHE. Tail of male×80.
7. *Subulura suctoria* Male & female Natural size.
" 8. *Subulura allodapa* Mouth×165.
9. *Subulura suctoria* Mouth×165.
" 10. *Subulnra acutissima* Mouth in depth×165.
" 11. *Subulura acutissima* Mouth surface×165.

Plate 5.

Fig. 1. *Subulura jacchi* TRAVASSOS.
2. *Subulura strongylina* TRAVASSOS. Material from Cryptnrus.
3. *Subulura otolicni* VAN BENEDEN. Anterior extremity.
4. *Subulura otolicni* Tail of female invagined.
5. *Subulura otolicni* Accessory piece.
6. *Subulura otolicni* Male & female. B Natural size.
7. *Subulura otolicni* Tail of male.
8. *Subulura otolicni* ovijector.
" 9. *Subulura otolicni* Tail of female.
10. *Subulura andersoni* COBBOLD. Tail of male 25 diam.
" 11. *Subulura andersoni* Tail of female. 25 diam.

Plate 6.

Fig. 1. *Subulura sarasinorum* MEYER. Cephalic extremity.
" 2. *Subulura sarasinorum* Tail of male.
3. *Subulura sarasinorum* Transversal section of lateral expansion.
4. to 9. *Subulura sarasinorum.* Transversal cuts of oesophagus an different heights.
" 10. *Subulura macronis* STEWART. Genital opening of male.
" 11. and 12. *Subulura macronis* Head of female×325.
13. and 14. *Subulura macronis* Tail of male×216 2/3.
" 15. Anterior extremity of female×75.
" 16. Tale of female×325.
" 17. Lateral membrane×750.

Plate 7.

Figs. 1 to 11. *Subulura macronis* STEWART. Transversal sections of body of male.

Plate 8.

Figs. 1 to 8. *Subulura leprincei* GENDRE.

Plate 9.

Figs. 1, 2 and 3. *Subulura elongata* SEURAT.
4. *Subulura noctuae* SEURAT.

Plate 10.

Figs. 1, 2 and 5. *Subulura seurati* SEURAT.
3 & 4. *Subulura leprincei* SEURAT.

Plate 11.

Figs. 1 to 4. *Subulura halli* SEURAT.

Plate 12.

Fig. 1. *Subulura suctoria.* Tail of male×75.
« 2. *Subulura suctoria.* Ovijector×140.
3. and 4. *Subulura strongylina*×75.
5. *Subulura differens*×75.

Plate 13.

Fig. 1. *Subulura allodapa.* Tail of male ×75.
2. *Subulura allodapa.* Head of male ×250.
« 3 & 4. *Subulura allodapa.* Ovijector×140.
5. *Subulura allodapa.* Tail of male 75.

Plate 14.

Fig. 1. *Subulura jacchi.* Larva×250.
« 2. Idem. Egg×250.
3. Idem. Ovijector×140.
4. & 5. *Subulura jacchi.* Tail of male×75.

Plate 15.

Fig. 1. *Subulura forcipata.* Tail of male ×75.
2. Idem. Ovijector×140.
3. *Subulura trogoni.* Head×250.
4. Idem. Tail of male×75.

Plate 16.

Fig. 1. *Subulura travassosi.* Material from Bucco chacuru. Ovijector×140
2. & 3. Idem. Tail of male×75.
4. & 5. Idem Material from Bucco swainsoni. Tail of male×75.

Plate 17.

Fig. 1. *Subulura lutzi.* Head of male×250
« 2 & 3. *Subulura lutzi.* Tail of male 75.
« 4. *Subulura lutzi.* Ovijector×75.

Plate 18.

Fig. 1. *Subulura bentocruzi.* Material from Trogon sp. Tail of male×75.
2. & 3. *Subulura bentocruzi.* Material from Trogon variegatus. Tail of male×75.

Plate 19.

Figs. 1, 3 & 4. *Subulura carlosi.* Tail of male×75.
2. *Subulura carlosi.* Ovijector×140.

Plate 20.

Fig. 1. *Subulura olympioi.* Material from Rhynchotus rufescens. Tail of male×75.
« 2. Idem, idem. Ovijector×140.
« 3. & 4. *Subulura olympioi.* Material from Nothura maculosa. Tail of male×75.
5. *Subulura olympioi.* Material from Crypturus parvirostris. Tail o male×75.

Plate 21.

Figs. 1 to 6. *Oxynema boueti* GENDRE.
7. *Oxynema crassispiculum* LINSTOW.

Plate 22.

Figs. 1 & 2. *Cissophyllus laverani* RAILLIET & HENRY. Mouth× 150.
3. *Cissophyllus laverani.* Tail of male×18.

Plate 23.

Fig. 1. *Heteroxynema cucullatum* HALL Male.
« 2. Idem. Female.
3. Idem Head, dorsal surface.

Fig 4. Idem Head, dorsal surface.
« 5. Sucker of male. Idem.
« 6. Idem. Tail of male.
7. Idem. Vulva×92,5.
8. Idem. Eggs×340.

Plate 24.

Fig. 1. *Numidica numidica* SEURAT. Head.
« 2. *Numidica numidica* Tail of male.
« 3. Idem. Ovijector.
« 4. Idem. Tail of male.

Contributions à l'étude de la faune helminthologique du Brésil

par le

DR. LAURO TRAVASSOS.

VIII

Les espèces brésiliennes du genre Tetrameres CREPLIN, 1846.

(Planches 25–28.)

Dans ma première contribution sur ce groupe originel de parasites, en 1914, j'ai donné une description du genre et des 4 espèces jusqu'alors connues au Brésil et un catalogue des synonymes et des hôtes de toutes les espèces du genre. Je donnerai à présent une liste des espèces, tout en les distribuant en deux sous-genres comme je l'avais proposé dans un autre article publié en Octobre 1915; en faisant mention seulement des synonymes et des hôtes dont je n'avais pas parlé antérieurement et j'étudierai les 7 espèces que nous avons tout récemment décrites.

Dans mon article antérieur, je décrivis une espèce comme *T. fissispina*. j'ai constaté depuis qu'il s'agissait d'une espèce nouvelle pour laquelle j'ai proposé le nom de *T. confusa* dans une note présentée à la *Primeira Conferencia Nacional de Pecuaria.*

Dans ce même article, je décrivis une autre espèce, qui infecte les canards comme *T. fissispina* et a été confondue par quelques auteurs avec la dite espèce.

Malheureusement, jusqu'à présent je n'ai pas pu obtenir le *T. paradoxa*, mais il me semble que les études de V. DRASCHE suffisent pour pouvoir établir les caractéristiques du genre et des deux sous-genres en lesquelles j'ai divisé ce groupe de parasites.

J'appelle l'attention des lecteurs sur le fait curieux qu'au Brésil l'on trouve *T. confusa* très fréquemment chez les l'oiseaux domestiques. WHARTON a observé le même fait aux Iles Philippines.

QUARTIM BARBOSA a aussi étudié ce parasite au points de vue pathogénique et économique dans sa thèse inaugurale.

Dans ma premiére contribution, j'ai considéré la possibilité de l'identité du parasite des poules au Brésil avec celui de *l'Anas* en Europe, ce qui était dû aux déscriptions insuffisantes des mâles et à la ressemblance des organes génitaux des femelles

dont, du reste, les caractéristiques sont comme ceux des *T. fissispina*, *confusa* et *gigas*.

Lors de mes dernières recherches j'ai eu l'occasion de voir des femelles vierges dont l'habitat était celui du mâle, c'est-à-dire le lumen du ventricule, ce qui fait croire qu'elles recherchent les glandes de cet organe après la copulation, en quoi ils ressembleraient à *Sarcopsylla penetrans*.

Quand il a pénétré la glande, le parasite tient son extrémité céphalique dirigée vers le fond de celle ci (fig. 7), d'où évidemment lui vient sa nourriture; il insère la partie coniforme entre les cellules de l'épithélium glandulaire; son extrémité est dirigée en dehors et se trouve dans l'ouverture glandulaire (fig. 8), pour que les oeufs puissent facilement être éliminés. Je n'ai rien pu déterminer quant à l'évolution de ce parasite; selon les observations de SEURAT, elle doit se faire dans un milieu liquide ou au moins humide. Je ne puis pas expliquer comment il arrive chez le nouvel hôte sans l'aide d'un hôte intermédiaire, parce que la plupart de ces oiseaux infectés par les parasites sont terrestres; les hôtes de *T. cruzi* et *minima* habitent les grands arbres et se nourrissent d'arthropodes et de fruits.

J'ai obtenu des cultures de larves de *T. confusa*, qui se sont conservées, pendant quelque temps dans les oeufs et qui furent seulement par hasard mises en liberté.

Des hôtes de *T. paradoxa* dont nous avons parlé dans notre article antérieur, il n'y a que *Catharistes atratus* qui doit entrer en considération.

Comme je l'ai proposé dans une note antérieure, j'ai divisé le genre Tetrameres en deux sous-genres, auxquels il faudra ajouter un troisième, si les recherches de SEURAT à l'égard de *T. gynaecophila* sont corroborées. Il nous semble qu'il y a eu quelque méprise de sa part: nous croyons que la forme décrite par lui comme mâle, soit une femelle très-jeune sans vestige de l'appareil génital; la forme très-curieuse du bout de l'intestin nous porte à soutenir notre opinion.

Tetrameres Tetrameres CREPLIN, 1846.

Tetrameres a spicules bien en proportion au corps, inégaux; le plus grand est de 3 à 6 fois plus long que le plus petit; la cuticle est garnie de épines, rangés latéralement sur les champs latéraux. Espèce type *T. Tetrameres paradoxa* DIESING 1834 TRAVASSOS 1914.

Tetrameres (Microtetrameres) TRAVASSOS, 1915.

Tetrameres à spicules inégaux; le plus grand est mal proportionné à la grandeur du corps (environ 2/3 de la longueur de celui-ci], le plus petit est peu chitinisé; champs longitudinaux sans cils; les femelles sont très souvent tordues sur l'axe longitudinal.

Espèce-type: *T. (Microtetrameres) cruzi* TRAVASSOS, 1914.

Liste des espèces du genre Tetrameres.

Sous-genre (Tetrameres).

1. T. (T.) paradoxa (DIESING, 1885) TRAVASSOS, 1914.

Sin.: *T. (Tetrameres) paradoxa* TRAVASSOS 1915.
T. (Tetrameres) paradoxa TRAVASSOS, 1917.
T. (Tetrameres) paradoxa BARBOSA 1917.

2. T. (T.) fissispina (DIESING, 1860) TRAVASSOS, 1914.

Sin: *T. (T). fissispina* TRAVASSOS, 1915 pr. part.
T. (T.) fissispina TRAVASSOS, 1917.
T. (T.) fissispina BARBOSA, 1917.
Hôtes.: *Cygnus melanocoryphus*.
Anas boschas.

3. T. (T.) nouveli (SEURAT, 1914) TRAVASSOS, 1914.

Sin.: *T. (T) nouveli* TRAVASSOS, 1915.

4. T. (T.) micropenis TRAVASSOS, 1915.

Sin.: *T. (Tetrameres) micropenis* TRAVASSOS, 1915.
T. (Tetrameres) micropenis BARBOSA 1917.
Hôtes:
Nyctanassa violacea L.

5. T. (T.) tetrica TRAVASSOS, 1917.

Sin: *T. (T). tetrica* TRAVASSOS, 1917.
T. (T). tetrica BARBOSA, 1917.
Hôtes: *Aramides cajanea* MUELL.

6. T. (T.) dubia TRAVASSOS, 1917.

Sin.: *T. (T). dubia* TRAVASSOS 1917.
T. (T.) dubia BARBOSA, 1917.
Hôtes.: *Gallinago paraguayae* VIEILL.

7. T. (T.) confusa TRAVASSOS, 1917.

Sin.: *T. fissispina* TRAVASSOS, 1914.
T. (T). fissispina TRAVASSOS, 1915 pr. part.
T. (T). confusa TRAVASSOS, 1917.
T. (T.) confusa BARBOSA, 1917.
Hôtes.: *Gallus domesticus.*
Meleagris gallopavo.
Columba livia dom.

8. T. (T.) gigas TRAVASSOS, 1917.

Sin: *Tropidocerca inflata* ZUERN, 1882, nec DIESING, 1860.
Tetrameres gigas TRAVASSOS, 1917.
Tetrameres gigas BARBOSA, 1917.
Hôtes.: *Anas boschas.*

Sous-Genre (Microtetrameres)

1. T. (M.) cruzi TRAVASSOS, 1714.

Sin.: *T. (M). cruzi* TRAVASSOS, 1915.
T. (M). cruzi BARBOSA, 1917.

2. T. (M.) inflata (DIESING, 1860) TRAVASSOS, 1914.

Sin: *T. (M). inflata* TRAVASSOS 1915.

3. T. (T.) inermis (v. LINSTOW, 1879) TRAVASSOS, 1914.

Sin: *Tropidocerca inermis* SEURAT, 1913
Tetrameres inermis TRAVASSOS 1915
Hôtes: *Corvus corax tingitanus* ISBY.
Passer domesticus (L.)
Lanius sp. (Pie griéche à tête rouge).

4. T. (M.) contorta (WIEDMANN 1913) TRAVASSOS, 1914.

Sin: *T. (M). contorta* TRAVASSOS 1915

5. T. (M.) minima TRAVASSOS 1915.

Sin: *T. (M). minima* TRAVASSOS 1915
T. (M). minima BARBOSA 1917.

6. T. (M.) spiralis (SEURAT, 1915) TRAVASSOS, 1915.

Sin: *Tropidocerca spiralis* SEURAT 1915
Tetrameres (M). spiralis TRAVASSOS, 1915.
Hôtes.: *Bubulcus lucidus* RAF.

7. T. (M). pusilla TRAVASSOS, 1915.

Sin.: *T. (M). pusilla* TRAVASSOS, 1915.
T. (M.) pusilla BARBOSA 1917.
Hôtes: *Turdus rufiventris* L.
Platycichla flavipes VIEILL.

Sous-Genre Indécis.

Tetrameres gynaecophila (MOLIN, 1858) TRAVASSOS, 1914.

Sin: *T. gynaecophila* TRAVASSOS 1915

2. T. unispina (DIESING, 1860) TRAVASSOS 1914.

Sin: *T. unispina* TRAVASSOS, 1915.

3. T. bispinosa (MOLIN, 1860) TRAVASSOS, 1914.

Sin: *T. bispinosa* TRAVASSOS, 1915.

4. T. globosa (v. LINSTOW, 1879) TRAVASSOS, 1915.

Sin.: *T. globosa* TRAVASSOS, 1915.

5. T. certa (LEIDY, 1880) TRAVASSOS, 1914.

Sin.: *T. certa* TRAVASSOS, 1914.

6. T. coccinea (SEURAT, 1914) TRAVASSOS, 1915.

Sin.: *T. coccinea* TRAVASSOS, 1915.

7. T. cochleariae TRAVASSOS, 1917.

Sin.: *Tetr. (T). micropenis* TRAVASSOS 1915.
T. cochleariae TRAVASSOS 1917.
Tetr. T. cochleariae BARBOSA. 1917.

De ces espèces on trouve au Brésil: *T. (T.) paradoxa, T. (T.) confusa, T. (M.) cruzi, T. (M.) minima*, que j'ai étudiées dans un travail antérieur et *T. (T.) fissispina, T. (T.) gigas, T. (T.) micropenis, T. (T.) tetrica, T. (T.) dubia, T. T. pusilla et T. cochleariae* que je décris ici.

Tetrameres (Tetrameres) fissispina (DIESING, 1860.)

(Planche XXVIII, figs. 9—11)

Femelle. Longueur 2,5 a 3 mm.; largeur 1 a 2 mm.

Capsule bucale, avec 0,021 mm. de profondeur sur 0,010 mm. de largeur, et la forme d'un fût; pharynx 0.23 mm. long, collier nerveux plus ou moins au milieu du pharynx; l'oesophage est environ 1 mm. long sur 0,087 mm. de largeur maxima; l'intestin a la forme d'un sac; l'anus se trouve à 0,071 mm. de l'extrémité postérieure; la vulve se trouve a 0,31 de l'extrémité; l'ovijecteur a un diverticule en forme de sac à sa partie initiale; les utéri sont très-longs et contiennent beaucoup d'oeufs; ils s'insèrent sur un vestibule très-long situé en avant de l'ovijecteur; à la partie terminale ils présentent des vésicules séminales; les ovaires sont très-longues et fusiformes; à la ponte enbryons développés) les oeufs ont environ 0,050 mm. de longs et 0,028 mm. de largeur.

Mâle: Longueur: 4 à 5 mm. Largeur: 0,14 mm. Le corps est filiforme, blanc; la cuticule, garnie de 4 rangs longitudinaux d'épines, rangées a côté des champs latéraux, est finement striée; papilles cervicales à 0,15 mm. de l'extrémité antérieure; bouche à deux lèvres peu visibles et une armature chitineuse qui se dirige en arrière jusqu'a 0,085 mm. de l'extrémité, accompagnant les expansions latérales; capsule buccale cylindrique, profonde, à environ 0,008 mm., de profondeur sur 10,003 mm. de largeur. Oesophage long de 0,78 mm. et large de 0,052 mm. (largeur maxima); intestin grêle; anus à 0,13 mm. de l'extrémité postérieure; 5 paires de papilles post-anales et une paire pré-anale, les épines de la cuticule formant un groupe de 3 a côté des papilles pré-anales; les spicules sont inégaux et mesurent respectivement 0,082 et 0,49 mm. en longueur; le plus petit est large de 0,007 mm. et uniformement cylindrique, le plus grand est large de 0,010 mm. et forme un angle a 0,14 mm. de l'extrémité proximale; il a un processus falciforme terminal, large de 0,050 mm.

Habitat: Ventricule de l'*Anas boschas.*

Cette espèce a ètè trouvée une seule fois, associée au *T. gigas.*

Tetrameres (Tetrameres) micropenis TRAVASSOS, 1915.

(Planche XXV, lfigs. 1 et 2; Pl. XXVII, figs. 7 et 8.)

Femelle: Longueur de 3 à 4 mm.; largeur de 1,5 à 2 mm.; corps rouge, globulaire, avec une forte striation transversale et 4 sillons profonds, qui correspondent aux champs latéraux et médians. Le collier nerveux se trouve à environ 0,18 mm. de l'extrémité buccale. La capsule buccale est ovoide, la profondeur en est de 0,02 mm. et la plus grande largeur de 0,014 mm.; la longueur du pharynx est environ 0,25 mm. et sa plus forte largeur 0,078; il est de plus en plus enflé en arrière; l'oesophage est plus ou moins cylindrique, la longueur est d'environ 1,5 mm. et la largeur au milieu 0,12 mm. La vulve se trouve peu au-dessus de l'anus; l'ovijecteur est simple et fort, claviforme; il a des muscles longitudinaux qui le font paraître strié (fig. 2]; la longueur en est environ de 0,71 mm.; l'utérus est double et long; les oeufs sont ellipsoïdes; ils ont

0,059 a 0,063 mm. de longueur pour 0,03 mm. de la largueur maxima, montrant des embryons déjà dans l'utérus.

Mâle: Longueur 4 à 5 mm. de largeur 0,12 la cuticule est délicatement striée en sens transversal et garnie de nombreuses épines dans les champs latéraux; deux paires de ces épines se trouvent au-dessous de l'anus et deux paires post-anales sur la surface ventrale (fig. 1.); les épines commencent à plus ou moins 0,35 mm. de l'extrémité antérieure; elles sont coniques, tournés en arrière et diminuent en grandeur vers l'extrémité postérieure; les antérieures mesurent 0,020 mm. en longueur sur 0,004 de diamètre à la base. Le collier nerveux se trouve à 0,191 a 0,198 mm. de l'extrémité antérieure; la capsule buccale est infundibuliforme a 0.028 mm. de profondeur le pharynx est 0,5 mm. long, l'oesophage presque cylindrique et a 1,3 mm. de longueur et 0,049 mm. à la plus forte largeur; l'anus se trouve a 0,184 mm. de l'extrémité; la queue est effilée; les spicules, inégaux, rélativement petits et effilés, mesurant respectivement 0,355 et 0,056 mm. en longueur.

J'ai trouvé dans le lumen du ventricule des femelles très jeunes avec les caractères suivants:

Longueur 2 mm.; plus grande largeur 0.22 mm.; corps strié transversalement, plus gros au niveau de l'oesophage à partir d'où il s'amincit jusqu'a l'extrémité postérieure; capsule buccale 0,021 mm. en profundeur et 0,007 mm. en largeur, cylindrique. Collier nerveux à 0,14 mm. de l'extrémite antérieure. Le pharynx a 0,27 mm. de longueur; l'oesophage a 0,60 mm. de longeur, l'anus se trouve à 0,16 mm. de l'extrémité postérieure.

Habitat: Ventricule de *Nyctanassa violacea* (L.): les mâles et les femelles vierges dans le lumen de l'organe, les femelles fécondées dans les glandes de LIEBERKUEHN.

Provenance: Manguinhos, Rio de Janeiro. Cette espèce est assez commune dans les socós des bourbiers de Manguinhos où nous l'avons trouvé.

Tetrameres (Tetrameres) tetrica TRAVASSOS, 1917.

Femelle: environ 1,5 mm. de longueur et 1 mm. de largeur; corps, rouge, presque rond et transversalement strié, la striation étant large et profonde comme dans les autres espèces: elle a 4 sillons profonds qui correspondent aux champs médians et longitudinaux; la capsule buccale ovoïde a les parois très épaisses, elle mesure 0,016 mm. en profondeur sur 0,012 mm. en largeur maxima. L'utérus termine postérieurement en une grande vésicule séminale ronde, qui a 0.10 mm. de diamètre; l'ovijecteur ressemble à celui de l'espèce sus-décrite; les oeufs ont 0,07 a 0,08 mm. de longueur sur 0,021 mm. de largeur; ils sont ellipsoides.

Mâles: Ils sont environ 2,6 mm. de longueur et 0,13 a 0,14 mm. de largeur maxima; le corps est plus gros au niveau de l'oesophage, d'ou il s'effile jusq'aux extrémités: la cuticle est transversalement striée, avec les striations très-étroites et il y a une quantité d'épines a côté des régions latérales; les épines de la cuticle commencent à 0,024 mm. de l'extrémité antérieure, où le corps est plus large; elles diminuent lentement et progressivement vers l'extrémité postérieure jusq'au dernier quart du corps; là elles disparaissent pour réapparaitre un peu en avant de l'anus; sur la surface ventrale il y en a 6, 4 paires sont post-anales et latérales il y a encore 4 paires sur la surface de la queue: les épines antérieures ont une longeur de 0,020 mm. et environ 0,003 mm. de plus fort diamètre. Le collier nerveux se trouve à 0,14 mm. de l'extremité antérieure. La capsule buccale irrégulière a environ 0,012 mm. de profondeur et 0,006 mm. de largeur; le pharynx cylindrique a 0,23 mm. de longueur; l'oesophage souscylindrique 0,51 mm. de longueur pour 0,042 mm. de largeur; l'anus se trouve à 0,20 mm. de l'extrémité postérieure qui est très pointue. Les spicules ont des dimensions très-differentes, le plus grand mesure 0,20 mm. en longeur et 0,006 en largueur et le plus petit, qui est très-peu chitinisé, a environ 0,022 mm. de longueur et 0,004 mm. de largeur

Habitat: Ventricule de l'*Aramides cajanea,* (MUELL.)

Provenance: Angra dos Reis (État de Rio de Janeiro).

Tetrameres (Tetrameres) dubia TRAVASSOS, 1917.

(Planche XXVI, fig. 3.)

Femelle: Longueur environ 2 à 2,5 mm. largeur 1 a 1,55 mm. le corps est jaune. avec striation transversale peu accentuée et 4 sillons longitudinaux profonds le long des champs latéraux et médians. La capsule buccale en forme de fût a 0,016 mm. en profondeur, pour 0,007 mm. de largeur maxima; l'anus se trouve à environ 0,075 mm. de l'extrémité. L'ovijecteur est du type de celui de *T. micropenis;* le spécimen examiné par moi était plein d'oeufs dans la partie dilatée ou il mesurait plus ou moins 0,42 mm. en longueur; les oeufs avaient environ 0,049 mm. de longueur sur 0,035 mm. de largeur maxima. Dans les spécimens examinés, les oeufs présentaient des touffes de cils aux pôles, ce qui a été aussi observé chez une espèce exotique, mais puisqu'on ne les trouve pas toujours et que les oeufs mûrs ne montrent pas un vestige de cette formation, nous ne savons à quoi attribuer leur l'origine; malheureusement nous n'avons pu examiner des specimens en état de fraîcheur, ni avec un fort grossissement mais il nous semble quand même que les cils doivent être d'origine parasitaire.

Mâle: Longueur environ 1,6 mm. sur 0,085 mm. de largeur maxima; le corps est effilé aux extrémités et plus large au niveau de l'oesophage; la cuticule est transversalement striée et présente de petites épines peu visibles. La capsule buccale, très-petite et plate, a environ 0,006 mm. de longueur sur 0,004 mm. de largeur; elle est presque carrée; le pharynx, presqu'aussi long que l'oesophage, mesure 0,24 mm. en longueur; l'oesophage mesure à-peu-près 0,30 mm. en longueur; l'anus se trouve a 0,10 mm. de l'extrémité postérieure; queue pointue, à 4 paires de petites épines sur la surface ventrale (fig. 5). Les spicules ont des dimensions très-différentes, mesurant respectivement 0,72 mm. de longueur sur 0,006 mm. de largeur et 0,004 mm. de longueur sur 0,004 mm. de largeur.

Habitat: Ventricule de *Gallinago paraguayae* (VIELL.)

Cette espèce représente un terme de passage entre les deux sous-genres que nous avons proposés, parceque, malgré la présence d'épines cuticulaires, (peu nombreuses du reste) le plus grand spicule a des dimensions intermédiaires entre les dimensions de ceux des deux groupes.

Tetrameres (Tetrameres) gigas TRAVASSOS, 1917.

Femelle: Longueur 5 à 6 mm. Largeur: 4 a 5 mm. Corps arrondi, jaune; cuticule transversalement striée, principalement au milieu; champs latéraux et médianes abaissées, formant ainsi 4 sillons longitudinaux. L'ovijecteur présente un diverticule en forme de sac à la partie initiale comme *T. fissispina* et *confusa*; les oeufs dont l'embryon est complètement développé à la ponte, ont 0,050 mm. de longueur sur 0,021 mm. de largeur.

Mâle: Longueur 7,5 mm., largeur: 0,18 mm. Corps filiforme, blanc, délicatement strié et avec 4 rangs longitudinaux d'épines, rangées latéralement à coté des champs latéraux. Le collier nerveux se trouve juste au-dessous du milieu du pharynx; les papilles cervicales se trouvent a 0,21 mm. de l'extrémité antérieure; le pore excréteur à 0,30 mm. La bouche a deux lèvres; la capsule buccale cylindrique a 0,021 mm. de profondeur sur 0,014 mm. da largeur, le pharynx 0,37 mm. de longueur; l'oesophage a 0,95 mm. de longueur et 0,087 mm. de largeur maxima; l'intestin est mince, l'anus se trouve à 0,12 mm. de l'extrémité postérieure; la queue est courbée vers la surface dorsale; les papilles manquent ou sont très peu visibles; les spicules diffèrent: le plus petit est très-réduit et ne mesure que 0,016 mm. en longueur, le plus grand mesure 0,74 en longueur et 0,014 mm. en largeur; l'angulosité basale se trouve

à 0,071 mm. de l'extrémité proximale, l'extrémité distale termine en pointe aigue.

Habitat: Ventricule de l'*Anas boschas*.

Provenance: Rio de Janeiro.

Tetrameres cochleariæ TRAVASSOS, 1917.

(Planche XXVII, 6),

Femelle: Longueur environ 3 à 4 mm. sur 1,5 mm. de largeur maxima; le corps est rougé en son aspect caractéristique; la capsule buccale a la forme d'un fût; elle mesure environ 0,024 à 0,028 mm. de profondeur sur 0,014 a 0,016 mm. de largeur; le pharynx a environ 0,19 mm. de longeur; l'oesophage, légèrement claviforme, a environ 0,96 mm. de longeur. Le collier nerveux se trouve à 0,14 mm. de l'extrémité antérieure: l'ovijecteur est très-long et musculaire (fig. 6), le vestibule et la partie proximale s'insèrent dans la partie terminale, faisant ainsi un coude; l'ovijecteur a environ 1,9 mm. de longueur sur 0,12 de largeur maxima; la largeur minima est de 0,05 mm. à la partie distale. Les oeufs sont ellipsoides, légèrement déprimés sur un des côtés; ils ont 0,042 de longueur sur 0,021 mm. de largeur maxima.

Mâle: inconnu.

Habitat: Ventricule de *Cancroma cochlearíae* L.

Provenance: Angra dos Reis, (État de Rio de Janeiro).

Nous n'avons pu obtenir des mâles de cette espèce qui parait assez fréquente chez ses hôtes, car nous avons trouvé des specimens deux fois sur trois; l'oiseau est cependant assez rare dans cette région.

Tetrameres (Microtetrameres) pusilla TRAVASSOS 1915.

(Planche XXV. fig. 3).

Femelle: Longueur 2 mm.; corps arrondi, rouge, à fortes stries transversales et 4 sillons longitudinaux dans les champs, latéraux et médians. Le collier nerveux se trouve à 0,092 mm. de l'extrémité antérieure; la capsule bucale, lagéniforme, a environ 0,010 a 0,16 mm. de profondeur sur 0,009 mm. a 0,012 mm. de largeur maxima; le pharynx a environ 0,21 mm. de longueur, l'oesophage a 0,57 mm. de longueur sur 0,09 mm. de largeur maxima; l'anus se trouve à 0,14 mm. de l'extrémité postérieure. La vulve est peu au dessus de l'anus; les oeufs sont ellipsoides, mesurent environ 0,042 a 0,049 mm. de longueur sur 0,028 a 0,035 mm. de largeur maxima et contiennent des embryons déjà dans l'utérus, comme d'ailleurs toutes les autres espèces de ce genre.

Mâle: Longueur environ 3,5 a 4 mm. sur 0,12 de largeur; le corps est mince; la cuticule est transversalement striée et ne présente pas d'épines; la capsule buccale est cylindrique à 0,017 mm. de profondeur sur 0,007 de largeur; le pharynx a 0,30 mm. de longueur; l'oesophage est pigmenté en jaune et légèrement claviforme; il a 0,042 mm. de longueur; l'anus se trouve a 0,17 mm. de l'extrémité caudale; l'extrémité postérieure a 5 paires de papilles asymétriques, dont deux paires sont pré-anales, une ad-anale et deux post-anales; les spicules ont des dimensions très-différentes; le plus grand a environ 1,32 mm. de longueur et 0,007 de largeur; l'extrémité proximale est légèrement dilatée et la distale arrondie; le plus petit mesure environ 0,085 mm. en longueur et 0,005 em largeur.

Habitat: Ventricule de *Turdus rufiventris* et *Platycichla flavipes* VIELL; les femelles dans les glandes de LIEBERKUEHN et les mâles et les femelles vierges dans la cavité de l'organe.

Provenance: Angra dos Reis, (État de Rio de Janeiro).

Literature.

BARBOSA, QUARTIM 1917 - Gastro-helmintose das aves domesticas. These inaugural, 1917. Rio de Janeiro.

FOSTER 1914 – A peculiar morphologic development of an egg of the genus Tropidocerca and its probable significance. J. Parasit, T. 1, pp., 45–47.

GROSSO, G 1914 – Ueber die Tropidocerca fissispina im Vormagen der Ente. Centr. f. Bact. T. 71, p. 271.

SEURAT 1913 – Observations sur la Tropidocerca inermis LINST. Bull. Soc. Hist. nat. Afrique du Nord T. 5, No. 8, p. 1, fig. 1–11.

SEURAT. 1915 – Sur deux Tropidocerca des Ardeidae. C. R. Soc. Biologie. T. 78, p. 279, fig. 1–4.

TRAVASSOS. 1914 – Contribuições para o conhecimento da fauna helmintolojica brazileira, IV. Mem. Inst. Oswaldo Cruz, T. VI. fasc. III.

TRAVASSOS 1915 – Sobre as especies brazileiras do genero *Tetrameres*. Brazil-Medico, T. 29, No. 38.

TRAVASSOS 1917 – Tetrameridae brazileiras. Brazil-Medico T. 31, No. 8.

TRAVASSOS 1917 – Gastro – helmintose das aves domesticas. Trabalho apresentado a 1ª conferencia Nacional de Pecuaria.

WHARTON, L. D 1918 – Tetrameres fissispina (DIESING, 186) in Philippine chickens. The Ph. Agr. a For. v. 6, No. 9 pp. 271.

WHARTON, L. D 1918 – Notes on nematode parasites of Philippine birds. *Tetrameres fissispina* (DIESING, 1860) in Philippine chickens. J. of Sc. V. XIII, No. 5. pag. 219.

Explication des Planches 25—28.

Pl. XXV. figs. 1. *T. (T.) micropenis.* Queue du mâle.
« « figs. 2. *T. (T.) micropenis* ovijecteur, mi-schématique.
« figs. 3. *Tetra. (Micr.) pusilla*- mâle.
XXVI. figs. 4. *T. (T.) tetrica* mâle.
« « figs. 5. *T. (T.) dubia* mâle.
« XXVII. figs. *Tetr.* (?) *cochleariae* ovijecteur.
« « figs. 7 e 8. *T. (T.) micropenis*, coupe du ventricule de *Nycta nassa violacea* où l'on voit la positon de la tête et de l'anus vis-à-vis de l'ouverture de la glande.
« XXVIII. figs. 9. *T. (T.) fissispina*- queue du mâle, face ventrale.
« figs. 10. *T. (T.) fissispina*- queue du mâle face latérale.
figs. 11. *T. (T.) fissispina*- tête, profil latéral.
« figs. 12. *T. (T.) gigas*, queue du mâle, face latérale.
figs. 13. *T. (T.) gigas*, tête, vue dorso-ventrale.
« « figs. 14. *T. (T.) gigas*, ovijecteur.

Encéphalite et Myélite causées par un trypanosome (T. Cruzi)

par les docteurs

MAGARINOS TORRES et JOÃO VILLAÇA

(Avec Planches 29-33).

Les altérations du système nerveux sont parfois nettement accusées et présentent des caractères spéciaux dans les maladies de l'homme causées par des protozoaires, telles que la syphilis, la maladie du sommeil et la malaria.

Dans la maladie de CHAGAS ou trypanosomose américaine, CHAGAS a signalée aussi une forme, ou il y a prédominance des troubles du système nerveux.

Cette maladie étant facile à réproduire artificiellement, il nous a paru intéréssant d'étudier les altérations du système nerveux central obtenues expérimentalement. Deux petits chiens furent inoculés par le *T. Cruzi*. L'étude histologique de leur système nerveux central révéla des lésions d'aspect très constant. Elles étaient de même nature chez les deux petits chiens et nous croyons devoir les ranger parmi les encéphalites et myélites, qualifiées par LOTMAR de: "procéssus bénins, aigus, accompagnés de prolifération de la névroglie du type des cellules à granulations. *(proliferierender, dem Körnchenzellen angehörigen Glia)*; c'est ici que l'on place l'encéphalite irritative et productive de FRIEDMAN et BONFIGLIO, ainsi que d'autres processus analogues de la pathologie humaine.

Matériaux et technique employée.

Chez les jeunes chiens l'inféction suit unet marche régulière et aboutit toujours à la mor. Comme nous avons eu l'occasion de constater au courant d'autres études, la myocardite se développe d'une façon remarquablement intense et constante chez ces animaux.

Les deux chiens, furent inoculées par voie souscutanée, avec du sang de cobaye contenant le *T. Cruzi*. L'un deux fût tué 18 jours après l'inoculation et l'autre mourut au bout de 25 jours.

Chien 64 nº. 1, agé d'un mois, inoculé sous la peau avec le T. Cruzi le 28 Janvier 1918.

Le 15 Février on trouve beaucoup de T. Cruzi dans le sang périphérique examiné.

On le tue, déjà agonisant, le même jour.

Chien 64 nº 3, âgé d'environ un mois, inoculé sous la peau avec le T. Cruzi, le 28 Janvier 1918.

L'examen du sang périphérique, le 15 Fevrier, montre un grand nombre de T. Cruzi.

Il meurt le 22 Février.

Le sang du coeur contient des T. Cruzi.

A l'autopsie des deux chiens on trouva les ganglions lymphatiques des régions axillaire et inguinale trés volumineux, la rate un peu agrandie, le coeur volumineux et flacide. Les circunvolutions cérébrales étaient nettes les sillons normaux. Les méninges et le liquide céphalo-rachidien étaient normaux. L'autopsie ne revèla pas de lésions grossiéres du tissu nerveux.

En résumé les lésions macroscopiques plus evidentes étaient celles du coeur et des ganglions lymphatiques.

Aucune lésion macroscopique du système nerveux central ou des meninges ne fut rencontrée.

Des fragments du cerveau, du cervelet, du bulbe, de la protubérance annulaire et de la moelle furent fixés á l'alcool á 95 o/o et au formol á 10 o/o. Les fragments fixés á l'alcool furent inclus au collodium. Les coupes feites au microtome á celloïdine furent colorées par la méthode de NISSL (bleu de toluidine).

D'autres fragments, inclus dans la paraffine, furent colorées par l'hématoxiline–éosine.

Description.

Les lésions observées étaient de même nature chez les deux chiens et ne présentaient que des variations d'intensité. Nous en donnerons donc une description d'ensemble, en signalant les particularités chez chaque individu.

Le système nerveux central présentait deux types de lésions dont les unes étaient bien marqueés et groupées en foyers et dont les autres peu intenses, étaient diffuses; ce sont a) les *lésions en foyer* et b) les *lésions diffuses*.

A coté de ces lésions, nous étudierons la distribution du *T. Cruzi* dans le système nerveux central, ainsi que les lésions que peuvent être attribuées à sa presence, ce que constitue une question bien importante.

A) Lésions en foyer.

Dans les coupes de cerveau et de moelle, colorées par la méthode de NISSL (bleu de toluidine) ces lésions sont perceptibles lorsque on les regard par transparence. Leur coloration n'est pas uniforme et l'on aperçoit des points, tant dans la substance blanche comme dans la substance grise, qui ont pris une coloration plus intense qne le tissu environnant (v. Pl. 29 et 30 figs. 1à 4).

Avec l'oculaire 2 et l'obejctif AA de ZEISS, ces lésions sont plus faciles à voir.

1e Distribution des foyers

Les lésions sont fréquemment trouvées dans des fragments quelconques de la moelle et du cerveau. Nous n'en trouvâmes aucune dans des coupes du cervelet. Dans la moelle et le cerveau on les trouve tant dans la substance grise comme dans la blanche, mais elles sont plus fréquentes dans cette dernière (Pl. 29 fig. 1 et 2).

Dans une coupe du cerveau du chien 64 n. 1 nous trouvâmes 36 foyers nettement limités, distribués de la manière suivante:

Substance grise. . . . . 14 foyers
Substance blanche. . . . . 22 foyers

Dans une coupe du cerveau du chien 64 n. 3, nous trouvâmes 13 foyers différents, avec la distribution qui suit:

Substance grise. . . . . 3 foyers
Substance blanche. . . . . 10 foyers

Dans les coupes de la moelle du chien 64 n. 1 les foyers étaient ainsi localisés:

Coupe n. I: 19 foyers; 1 dans la substance grise, 18 dans la blanche.

Coupe n. II: 20 foyers; 6 dans la substance grise, 14 dans la substance blanche.

Coupe n. III: 13 foyers; 3 dans la substance grise 7 dans la substance blanche et 3 dans le limite des deux.

Coupe n. IV: 7 foyers; 3 dans la substance grise, 4 dans la substance blanche.

Dans les coupes de la moelle du chien 64 n. 3 les foyers etaient ainsi repartis:

Coupe n. I: 13 foyers, 5 dans la région de séparation des deux substances et 8 dans la substance blanche.

Coupe n. II: 8 foyers, 1 dans la région de separation, 7 dans la substance blanche

Coupe n. III: 5 foyers, 2 dans la région de se-

paration, 3 dans la substance blanche.

Coupe n. IV : 3 foyers, tous dans la substance blanche.

Les dimensions des foyers varient. Le diametre des plus grands est d'environ 100 μ, et celui des plus petits d'environ 50 μ.

Quelquefois, assez rarement d'ailleurs, on trouve des foyers rapprochés; dans ce cas la région lesée présente des dimensions plus considérables.

2º Étude microscopique des foyers.

a) FOYERS DE MYÉLITE

Foyer A – Chien 64 nº. 3.

En examinant un foyer de myélite du chien 64 n. 3, à un fort grossissement (Oc. comp. 6. obj. d'imm. hom. 1/12 Zeiss), nous avons pu constater qu'il se compose de differentes sortes d'éléments cellulaires, dont nous allons décrire les caractères (v. Pl. 33 fig. 9).

On voit en premier lieu des nombreuses cellules à grand noyau arrondi, oval ou allongé et pauvre en chromatine. La chromatine se rencontre sous la forme de 2 à 3 masses irregulières plus grandes et nettement détachées de la menbrane cellulaire et de quelques granulations; l'on voit en plus une fine couche concentrique de chromatine déposée sur la membrane cellulaire qui est bien délimitée.

Le protoplasme est aussi nettement délimité et presente de 4 à 5 prolongements qui suivent des differentes directions et vont à la rencontre d'autres prolongements semblables issus des cellules voisines (v. Pl. 63, fig. 9). Nous considérons ces cellules comme des cellules de la névroglie qui ont proliféré et se sont ramifiées.

On remarque encore d'autres cellules, dont le noyau est plus petit, reguiér, rond et riche en chromatine qui prend la forme de nombreuses granulations, dont quelques unes sont liées à d'autres par des trabécules chromatiques. Quand on colore ces cellules au bleu le toluidine, leur suc nucléaire prend une teinte plus foncée que dans les cellules précédentes. Le protoplasme est bien delimité; dans beaucoup des cellules il contient des nombreuses granulations (v. Pl. 33, fig. 9).

Ces cellules présentent beaucoup des caractères signalés pour les *Abraümzellen* d'origine névroglique (LOTMAR). Nous remarquâmes en plus dans ces foyers des cellules de la névroglie, dans lesquelles les signes de regression étaient bien en évidence (noyaux en picnose, protoplasme réduit à des granulatitions, fragmenté).

Dans ces foyers il n'y avait pas de cellules plasmatiques ni d'autres éléments d'origine hematogène (polymorphonucléaires, lymphocytes).

On trouve parfois le *T. Cruzi* dans des foyers semblables; il prend alors la forme d'un corpuscule leishmaniforme (Pl. 30, fig. 5).

Dans les mêmes coupes de la moelle nous trouvâmes des foyers que different des précedents, par certains aspects.

Foyer B – Chien 64 nº. 3. (Pl. 31, fig. 6)

Les éléments qui forment ce foyer sont tres concentrés, de façon qu'il prend une coloration bien intense. Dans la partie centrale ont ne voit clairement que des noyaux qui, examinés rapidement, donnent l'impression d'être disséminés, au sein d'une masse protoplasmique intensement colorée. (Pl. 31, fig. 6). A la périphérie, les cellules de la névroglie sont plus éparses et leurs prolongements vont à la recontre d'autres, issus de cellules de même nature. Les noyaux en picnose sont assez nombreux. Il y a quelques rares cellules analogues aux "*Abräumzellen*" d'origine névroglique (LOTMAR).

Foyer C—Chien 64 n. 3.

Les cellules sont plus éparses, de façon qu'au faible grossissement la coloration est moins intense.

Les élemets cellulaires sont presqu'exclusivement des cellules de la névroglie, présentant des alterations d'ordre progréssif. Leur protoplasma a des limites moins précis que

celui des précédentes. Quelques unes sont en caryokinèse.

b) FOYERS d'ENCÉPHALITE.

Foyers D—Chien 64 nº. 3.

Les foyers d'encéphalite ont la même structure générale que les foyers de myélite étudiés chez les mêmes sujets. On voit des cellules de la névroglie en voie de prolifération, des *"Abraumzellen"* et des cellules de la névroglie altérées et degénérées (c'est á dire avec leur noyau em picnose et entouré de granulations); (comparer avec les figs. 3 *n*, *o*, *p* de la publication de ALZHEIMER[1]). En plus de ces éléments on voit des cellules plasmatiques.

Foyer E—Chien 64 nº 1

Ce foyer est formé par des cellules de la névroglie qui ont proliferé et par des cellules plasmatiques. Ces éléments sont de beaucoup plus nombreux que dans les foyers d' encéphalite du chien 64 nº 3, ce qui ne manque pas d'attirer l'attention. Les altérations du systeme nerveux central sont plus accusées chez le chien 64 nº 3. Tout semble démontrer que chez le chien 64 nº 1 les lésions sont plus anciennes (foyers d'encephalite et de myélite plus nombreux, présence de cellules plasmatiques en quantité plus grande, fréquence des amas de *T. Cruzi*, dont beaucoup affectent la forme flagéllée). Ce fait est tres intéréssant et mérite d'être signalé, puisque la durée de l'infection est moindre pour ce chien que pour le chien 64 nº 3.

De cette sorte, le systéme nerveux central serait attaqué plus vite et d'une façon plus intense chez certains animaux que chez d'autres, pour lesquels, quoique la durée de l'infection fut plus longue, les lésions des centres nerveux seraint bien moins intenses. On pourra peut-être voir une analogie entre ce fait et d'autres semblables, observées chez l'homme atteint de la maladie de CHAGAS.

B. Les lésions diffuses.

Tant dans les préparations de la moelle, contenant des foyers de myélite, comme dans celles du cerveau où les foyers d'encéphalite sont nombreux, on remarque que les cellules ont en général consevé leur structure, lorsqu'on les examine par le methode de NISSL. Elles se comportent donc d'une façon bien différente que dans le cas de la paralysie des chiens, dans laquelle les altérations sont accentués ou les cas d'intoxication par la toxine dysentérique (LOTMAR[7]) et par le carbonate de plomb (BONFIGLIO[8]).

Dans quelques points, qui sont d'ailleurs relativement rares, on voit des cellules de la névroglie disposées autour d'une cellule nerveuse qu'elles entourent presque; cette cellule montre de altérations de structure (vacuoles dans le protoplasma, dont le reste prend une coloration diffuse ne permettant pas de distinguer les granulations de NISSL).

Quelques unes des coupes de la moelle preséntent une prolifération vasculaire assez marquée.

Le T. Cruzi et les foyers d'encéphalite et de myélite.

Des agglomérations de *T. Cruzi* se font remarquer, non pas dans le foyer d'encéphalite des coupes du cerveau du chien 64 nº 1, mais dans leur voisinage (Pl. 32 fig 8 en haut à gauche). Ils se présentent alors sous la forme flagéllée, qui precède la forme typique du sang (G VIANNA). L'amas de trypanosomes est nettement limité par la membrane de la cellule envahie (Pl. 32 fig. 8.) et prend une forme sphérique, dont le diamètre moyen est de 42 μ étant déjà perceptible au faible grossissement. (Oc. comp. 6 et obj. AA ZEISS).

L'amas est entièrement composé d'organismes flagellés trés nombreux. Dans les préparations colorées au bleu de toluidine, on les voit sous la forme de bâtonnets, dont les extrémités sont éffilées et courbées en plusieurs plans. Il y a une remarquable difference de dimensions entre les différents individus, qui sont d'alleurs diffi-

ciles à observer, vu leur grand nombre et le fait que les uns recouvrent les autres, ou s'entrelacent avec eux. L'observation des menus détails est difficile par la méthode de NISSL. Le plasma des bâtonnets prend une teinte bleue violet pâle; à une des extrémités l'on aperçoit un point violet foncé qui correspond au blépharoplaste et au milieu une masse à limites mal définies, d'un bleu violet moins intense; c'est le noyau principal.

Nous tenons à signaler que ce n'est que rarement que l'on peut observer le noyau de la cellule envahie par ces kystes d'individus flagéllés. Nous l'attribuons au fait que les coupes n'intéréssent que rarement le noyau des cellules dilatées. Quand par hasard il est visible, il ressemble au noyau des cellules de la névroglie. (Pl. 33 fig. 10). Dans quelques unes des agglomerations, les flagéllés n'occupent que la partie centrale de la cellule envahie. (Pl. 32 fig. 8 en haut à gauet Pl. 31 fig. 7); autour de cette partie centrale, remplie de trypanosomes, on voit un halo clair, lui-même entouré par la membrane cellulaire. Il nous semble que cet aspect est dû à l'action de l'agent fixateur.

Dans des coupes de la moelle du chien 64 nº 1 et du cerveau du chien 64 nº 3, on trouve des cellules contenant des *T. Cruzi,* affectant la forme d'une Leishmania, dans les propres foyers. Ces cellules sont bien distinctes et en général très nombreuse (environ de 5 à 20); dans leur protoplasma on aperçoit deux masses nucléaires; une correspond au noyau principal et l'autre au blépharoplaste.

Étant donné les dimensions des foyers d'encéphalite et de myélite et n'ayant pas fait des coupes en série, il nous est impossible de dire quelque chose de définitif au sujet de la fréquence du *T. Cruzi* dans les mêmes.

Dans une des préparations du cerveau du chien 64 nº 1 nous trouvâmes 14 foyers dans la substance grise et 22 dans la substance blanche; aucun d'eux ne contenait le *T. Cruzi,* mais dans cette même coupe on voyait le *T. Cruzi* sous la forme d'amas d'individus flagéllés en plein tissu nerveux. Une autre coupe de cerveau du chien 64 nº 1 contenant 6 foyers d'encéphalite, dont 4 dans la substance grise et 2 dans la blanche, on pouvait voir des trypanosomes leishmaniformes dans un de ces foyers. Il est pourtant possible que nous eussions eu des résultats différents dans le cas de la prmière coupe, si nous eussions fait des coupes en série des 36 foyers qu'elle montrait.

Em résumé, nous dirons que le *T. Cruzi* affecte deux formes diverses dans le système nerveux central du chien. Tantôt on le trouve à l'intérieur d'une cellule situé au sein du tissu nerveux, loin de tout foyer d'encéphalite et de myélite, ainsi que des vaisseaux sanguins. La cellule envahie est alors tellement distendue qu'on l'aperçoit même à un faible grossissement. (Pl. 32, fig. 8 en haut, à gauche). Le trypanosome prend la forme d'un organisme flagéllé Les individus sont tellement amassés qu'il est difficile d'en distinguer un, isolément. (Pl. 31 fig, 7 et Pl. 33 fig. 10).

Dans l'autre cas la cellule envahie se trouve en plein foyer d'encéphalite ou de myléite ou dans son voisinage immédiat et ne contient en général que peu de trypanosomes (de 5 à 20) qui prennent toujours la forme d'une Leishmania.

Il est intéréssant de noter que chez le chien 64 nº 3 les trypanosomes furent fréquemment rencontrés dans les propres foyers d'encéphalite et de myélite, affectant la forme d'un Leishmania, tandis que dans le cerveau du chien 64 nº 1, où tout semble indiquer que le procéssus est plus ancien, les trypaosomes sont trouvés souvent à l'intérieur des cellules en dehors des foyers et de leur voisinage et qu'ils présentent alors leur forme flagéllée.

Du diagnostic différential.

Nous avons pu faire des obsérvations trés soigneés sur l'évolution de l'infection chez les animaux dont nous étudions le système nerveux central en ce moment. Puisque ces animaux sont sujets à bien d'autres

maladies, l'observation clinique s'imposait. Parmi ces maladies il faut ranger premièrement la maladie des jeunes chiens (*"canine distemper"*, *"staupe"*) Cette maladie produit, surtout quand elle se présente sous la forme nerveuse, des lésions considérables du système nerveux central qui ont été bien décrites par CERLETTTI[3] et DEL RIO HORTEGA[8].

a) L'OBSERVATION CLINIQUE.

Nous n'avons eu l'occasion de remarquer aucun symptome qui pourrait faire croire que nos chiens avaient la maladie des jeunes chiens. Nous n'observâmes a aucun moment le catarrhe des muqueses du nez, des yeux ou des voies respiratoires, ni la conjunctivite, la kératite, l'éruption cutanée ou les phenomènes d'ordre nerveux plus ou moins prononcés tels que vomissements, nystagmus, paralysie ou tremblements. Ils se porterent bien pendant toute la période de l'observations à l'exception seulement des 24 heures avant leur mort. D'un jour à l'autre ils refusèrent leur nourriture, commencèrent à agoniser et venaient à mourir dans les 24 heures.

En passant soit dit que nous attribuons leur mort à la myocardite constamment observée chez les jeunes chiens inoculés avec le *T. Cruzi.*

b). L'EXAMEN HISTOPATOLOGIQUE.

L'examen histo-pathologique permet de distinguer facilment entre les processus que nous venons de décrire et que l'on rencontre chez les jeunes chiens inoculés avec le *T. Cruzi* et les autres procéssus dont l'étiologie n'est pas la même.

Ici encore les plus importants sont la maladie des jeunes chiens et les trypanosomiases d'ordre expérimental (tabes du chien SPIELMEYER).

Les principales différences entre les processus étudiés par nous et ceux décrits par CERLETTI et RIO HORTEGA sont les suivantes:

1. Dans la maladie des jeunes chiens les lésions cérébrales sont diffuses et s'étendent sur tout le cortex cérébral.

Chez nos chiens les lésions sont surtout des lésions en foyer.

2. Dans la maladie des jeunes chiens elles sont plus intenses dans la substance grise. Les lésions des cellules nerveuses sont bien accusées, surtout dans la couche moléculaire et dans celle des petites cellules pyramidales.

Chez nos chiens les lésions sont plus acusées dans la substance blanche.

3. Dans la maladie des jeunes chiens les lésions du cervelet sont très intenses, même plus marquées que celles du cerveau; les lésions de la substance blanche, qui sont inflammatoires predominent.

Le cervelet de nos chiens ne présentait point de lésions perceptibles; on n'y trouvait pas non plus des foyers de prolifération de la névroglie, ni des cellules envahies par le *T. Cruzi.*

4. Dans la maladie des jeunes chiens l'infllammation des mêninges est um phénomène constant.

Chez nos chiens elle n'existait pas.

5. On trouvait chez nos chiens, au niveau du foyer et quelques fois en plein tissu nerveux, des cellules dont le plasma contenait des *T. Cruzi*, soi des corpusculles leishmaniformes, soit des formes flagéllés.

Chez des animaux inoculés avec le *T. Brucei*, SPIELMEYER constata une dégénération élective des racines postérieures de la moelle qui au point de vue histologique, ressemblaient beaucoup à la tabes.

Nous n'avons pas eu l'occasion d'employer la méthode de MARCHI ou une autre en usage pour les recherches sur la dégénération des faisceaux conducteurs.

SPIELMEYER ne décrit chez ses chiens rien que puisse rappeller la myélite observé chez les nôtres.

Conclusions.

1º. Les chiens inoculés avec le *T. Cruzi.* présentent des altérations du système nerveux central, qui peuvent être rangées parmi

les myélites et encéphalites, que LOTMAR décrit comme des processus bénins à marche aigue et accompagnées de prolifération des cellules à granulations.

2o. La myélite et l'encéphalite sont caractérisées par l'existence de nombreux foyers localisés de préférence dans la substance blanche de la moelle et du cerveau.

3o. Le foyers sont composés de cellules hypertrophiées de la névroglie, de cellules que nos considérons comme des *Abraumzellen* d'origine névroglique, de cellules de la névroglie présentant des phénomènes d'ordre regressif; on y trouve en plus, surtout dans les foyers localisés dans le cerveau, un nombre plus ou moins grand de cellules plasmatiques (*Plasmazellen*).

4o. Les cellules nerveuses du cortex cérébral et de la moelle conservent en général leur structure, même quand elles se trouvent dans la voisinage immédiat des foyers d'encéphalite et de myélite.

5o. Les meninges ne sont pas le siège d'inflammation. En quelques points, rares d'ailleurs, on voit une accumulation de cellules autour des vaisseaux de la pie-mère.

6o. Dans beaucoup de foyers d'encéphalite et de myélite et parfois en plein tissu nerveux, on trouve le *T. Cruzi;* il se présente alors soit sous la forme d'une Leishmania, soit sous la forme flagéllée.

7o. Il y a une relation évidente entre la morphologie du *T. Cruzi* et l'emplacement de la cellule envahie dans le tissu nerveux.

Dans la phase de corpuscule leishmaniforme, qui est d'ailleurs la première forme prise par le trypanosome du sang quand il se localise dans les tissus, la cellule envahie est une des celles qui se trouvent dans le foyer même d'encéphalite et myélite ou dans son voisinage immédiat.

Pendant la phase flagéllée, phase qui précede immédiatement le passage de ce parasite dans le sang, il occupe une cellule située au sein du tissu nerveux, loin de tout foyer d'encephalite et de myélite ou des vaisseaux sanguins.

8o. Il n'est par décrit dans ce travail, une paralysie des chiens causée par le *T. Cruzi.*

L'observation clinique ne nous montra pas des signes qui feraient songer aux lésions du systeme nerveux central. Ces observations, rapportés ailleurs, permetent d'affirmer que les chiens étaient en bonne santè appaenrte jusqu'auxdernières 24 heures qui précederènt la mort; due à la myocardite aigue comme on le vérifia par l'examen microscopique.

9o. Nous nous sommes éfforcés de décrire exatement les lésions histo-patologiques spécifiques et non spécifiques du système nerveux central des animaux en expérience, inoculés avec le *T. Cruzi,* de façon à permettre une connaissance plus détaillée de l'histologie du systeme nerveux dans la *trypanosomose de* CHAGAS.

En finissant, nous tenons à presenter nos remerciements au Dr. B. C. CROWELL qui a eu l'obligeance de lire notre manuscript et d'examiner nos préparations, en nous donnant des excellents conseils et à exprimer notre reconnaissance au Dr. CHAGAS auquel nous devons les possibilités de travail, ainsi que l'orientation des recherches.

Literature.

1 – ALZHEIMER A. – Beit. z. Kenntniss d. path. Neuroglia u. ihrer Beziehungen z. den Abbauvorgängen im Nervengew. Histol. u. histopath. Arb. über d. Grosshirnrinde u. s. w. herausgegeben von NISSL, F. und ALZHEIMER, A. Bd. III (1910) p. p. 401.

2 – BONFIGLIO, F. – Circa le alterazioni della corteccia cerebrale conseguenti ad intossicazione sperimentale da carbonato di piombo. (Encefalite produtiva). Histol. u. histopath. Arb. über d. Grosshirnrinde Bd. III, (1910) p. p. 359.

3 – CERLETTI, U. – Über verschiedene Encephalitis-und Myelitisformen bei an Staupe erkrankten Hunden. Zeitschr. f. d., gesamte Neurol. u. Psych. *Originalien* Bd. IX (1912 p.) p. 520.

4 – CHAGAS, C. – Nova entidade morbida do homem. Rezumo geral de estudos etiolojicos e clinicos. Mem. Inst. Oswaldo Cruz. T. VII, Fasc. 2 (1911) p. p. 219.

5 – CHAGAS, C. – Processos patojenicos da tripanozomiase americana. Mem. Inst. Oswaldo Cruz. T. VIII, Fasc. II, (1919) p. p. 5.

6 – CHAGAS, C. – Tripanozomiase americana forma aguda da doença. Mem. Inst. Oswaldo Cruz. T. VIII, Fasc. II, (1919) p. p. 37.

7 – LOTMAR, F. – Beitr. zur Histologie der akuten Myelitis u. Encephalitis sowie verwandter Prozesse. Histol. u. histopath. Arb. über d. Grosshirnrinde Bd. VI (1913) p. p. 245.

8 – RIO HORTEGA, P. D. – Alteraciones del sistema nervozo central en un caso de moquillo de forma paralitica. Trab. d. Lab. d. Inv. Biol. T. XII (1915) p. p. 97.

9 – VIANNA, G. – Contribuição para o estudo da anatomia patholojica da "Molestia de Carlos Chagas" (Esquizotripanoze humana ou tireoidite parazitaria). Mem. Inst. Oswaldo Cruz T. III, fasc. 2 (1911) p. p. 275.

Explication des Planches.

Pl. 28 Fig. 1

Chien 64 n. 1

Coupe de cerveau (cortex) Collodium. Bleu de Toluidine. Microphotographie.

Lésions groupées en foyers, localisés dans la substance blanche.

Pl. 29 Fig 2

Chien 64 n. 1

Coupe du cerveau (cortex). Collodium. Bleu de Toluidine. Microphotographie.

Foyers d'encéphalite (dont un à la séparation des couches corticale et médullaire).

Pl. 30 Fig. 3

Coupe transversale de la moelle. Collodium. Bleu de toluidine. Microphotographie.

Foyers de myélite localisés dans la substance blanche.

Pl. 30 Fig. 4

Chien inoculé avec le *T. Cruzi*.

Coupe longitudinale de la moelle. Microphotographie. Paraffine, hématoxyline de Hansen. Éosine.

Foyers de myélite localisés dans la tissu médullaire et cortical.

La microphotographie fut faite avec le objectif 2 mm. sans oculaire.

Pl. 30 Fig. 5

Chien 64 n. 1

Coupe de cerveau. Collodium. Bleu de toluidine. Microphotographie.

Foyer d'encéphalite, dans la partie centrale duquel on voit une cellule contenant le *T. cruzi* sous la forme de leishmania, (peu net dans la photographie).

Pl. 31 Fig. 6

Chien 64 n. 3

Coupe de la moelle. Collodium. Bleu toluidine. Microphotographie.

Foyer de myélite (le plus antérieur vu dans la fig. 3 Pl. 30) au fort grossissement.

Pl. 31 Fig. 7

Chien 64 n. 1

Partie de la coupe vue Pl. 32, au fort grossissement, montrant un amas de *T. cruzi*, a l'intérieur d'une cellule.

Pl. 32 Fig. 8

Chien 64 n. 1

Coupe de cerveau. Bleu de toluidine.

Grand amas de *T. cruzi*, forme flagéllée en plein tissu nerveux.

Pl. 33 Fig. 9

Coupe de la moelle. Collodium. Bleu de toluidine.

Foyer de myélite, décrit à la page 82 du texte.

Pl. 33 Fig. 10

Chien 64 n. 1

Cellule très grossie contenant le *T. cruzi* dans son protoplasma. La coupe intéresse le noyau de la cellule.

Experimental Beriberi and Beriberi in Man, with special reference to its forms, as observed in Amazonas

by

DR. FIGUEIREDO RODRIGUES.

(With Plates 34 – 36).

The etiology of beriberi is still a matter of controversy amongst us. The greatest divergence of opinion reigns with regard to many of its clinical phenomena. This being the case, any contribution, however modest, to the solution of this important problem of nosology is of the greatest interest. Consequently I thought that my cooperation in the analysis of the problem might be of some use, especially as the results given here are those of clinical observation and experimental work.

The disease is frequent in tropical regions, and takes a prominent place in the archives of our medical history, not only because it wrought havoc amongst our soldiers in the Paraguay campaign, but as it still causes a great mortality on board our men-of-war and amongst the heroic pioneers in the uncultivated lands and fields in the north of Brazil. Therefore, I feel constrained to spare no trouble or painstaking in the study of this scourge.

It was unquestionably only after the epidemic in Bahia, in 1864, that the attention of medical men in the New World was called to beriberi. The foremost place of honour undoubtedly belongs to SILVA LIMA, one of the very first to study the disease from the clinical point of view. The name of SILVA LIMA must be mentioned with pride and esteem, as that of a pioneer in studying beriberi or even of its rediscoverer, 200 years after it had been mentioned as a Brazilian disease by WILLEM PISON in his work "De Medicina Braziliensi" published in Holland in 1864.

From the time of SILVA LIMA up to the present date, many of our compatriots have contributed to the study of the clinical symptoms of this disease, and TORRES HOMEM brings the value of Brazilian studies into relief, while making a special reference to the work of the great Bahian doctor.

I cannot pass over this interesting chapter of pathology, nor forget that it is my duty to take part in the debates which followed the experimental work of EIJKMAN. Such studies have thrown so much light upon the subject, and called forth so many magnificent experiments and observations in the Orient, that we might almost conclude that all problems were solved, as well from the clinical as from the experimen-

tal point of view. But there being still some reluctance amongst our most considered medical authorities to accept the conclusions drawn from these experiments in the East, I think the subject is actually of the highest interest, as well from the scientific as from the pratical point of view.

I have divided this work into three parts: 1. Beriberi in man and its symptomatology. 2. Experimental Beriberi and its relation to Beriberi in man. 3. Ætiology of Beriberi.

Beriberi in man and its symptoms.

All Brazilian authors define Beriberi by the discrimination of its symptoms and its etiological cause. SILVA LIMA describes the former as follows: "constitutional disease, endemic or epidemic, characterised by insensibility of the extremities, general weakness and inability to move, with pains and heaviness in the muscles, often accompanied by rigid oedema, anasarca, swelling of the face, anemia, epigastric oppression, dyspnoea (generally gradual), partial paralysis, sometimes accompanied by constriction round the trunk, weaknes and hoarseness; spasmodic movements of the limbs; in fatal cases the end survenes from asphyxia and extenuation; in the favorable ones, after abundant diuresis, a slow and gradual restoration to normal conditions sets in and the nervous and circulatory systems are regularised."

AZEVEDO SODRÉ defines it as "an infectious, probably contagious illness, becoming endemic or epidemic in tropical and sub-tropical regions, the characteristics of which are: a more or less accentued motor paralysis beginning with the lower extremities, accompanied by partial oedema and disturbances of the senses, and sometimes general dropsy; by visceral alterations to which special attention is called; by perturbations of the heart and exacerbations of the lungs: by chronic progress, sometimes interrupted by acute exacerbations which may give rise to aggravation of the symptoms already existing and to new ones; death may be caused by asphyxia or cardiac syncope".

MIGUEL COUTO also describes the symptoms without making a synthesis of them.

From the ideas which we shall expose presently and from the result of modern study of beriberi, we may say that it is a disease produced by insufficient alimentation, characterised by a generalised neuritis, attacking the sensory and motor nerves and also those dependant on the great sympathetic, thus provoking multiple visceral perturbations, those of the heart being pathognomic.

Prodromatic periods: According to what I observed in the province of the Amazonas, anorexy, is perhaps the initial phenomenon of beriberi; before the disturbances of sensibility and motility set in, the patients complain of want of appetite.

TORRES HOMEM, SILVA LIMA and SODRÉ think that in many cases the forerunners of the disease are melancholy, disinclination to work, fatigue arising from the slightest exercise, bad dreams, nightmare, sleeplessness, anorexy and palpitations. SODRÉ, PEKELHARING and WINKLER however affirm that if the muscles were examined by electrity before these symptoms set in, the characteristic marks of the disease would already be found; for them, galloping beriberi lasts longer than is generally supposed, the prodromatic symptoms passing unperceived.

In the state of Amazonas I saw cases of galloping beriberi, almost without prodromatic symptoms. It is as if the disease broke out suddenly, affecting the peripheral nerves as well as those of sensibility. I observed some superacute cases where the first and slighter perturbations (as for instance anorexy, general weakness, feeling of discomfort) lasted only three or four days, before the disease burst out with all its more serious symptoms.

PLEHN observed cases in Cameroon that developed fully in from twelve to forty-

eight hours. SILVA LIMA cites one that only lasted five days.

Nevertheless, we may affirm that the disease has generally a well characterised, distinct, initial and prodromatic state. This is ushered in by anorexy, as is also the case in experimental beriberi. Birds, before manifesting the typical signs of the disease, show almost complete anorexy. I noticed the same in the animals I experimented upon. ARLINDO de ASSIS also verified this fact and had to feed his artificially, to prevent them from dying of starvation, as he writes in his thesis which was suggested by Prof. CLEMENTINO FRAGA.

In the Malay peninsula, FRASER and STANTON also observed lack of appetite as an initial symptom of the disease in the labourers who furnished them whith matter for their classic experiments.

Besides anorexy some of the primary symptoms are:— fatigue after the least effort, palpitations and heaviness, a slight torpor of sensibility and weakness in the inferior extremities. These symptoms are described by all the authors on the subject. SAMPAIO VIANNA found a lack of appetite in 34o/o of the cases under his observation. I also noticed the appearence of vomiting in the beginning of the disease. This is a very bad sign. A sensation of fulness in the stomach often accompanies the want of appetite. The patients refuse to eat, because they feel as if their stomachs were full. In the north the patients have an expression of their own for this feeling of "stuffiness". I consider this symptom as almost constant. SAMPAIO VIANNA observed "fullness" in 27o/o of his cases.

The consequence of the alteration of the nerves of the stomach is gastric embarassment, and with this often comes fever.

Fever: According to my observations beriberi is an apyretic disease. Fever, when present, is a complication. SODRÉ is one of the modern authors who speak of a fever-temperature during the the course of the illness. He states that SARAIVA of Bahia called attention to the existence of fever in beriberi, when he circumstantially described the febrile forms observed in the plains of Paraguay. I believe that there was some confusion amongst the cases of beriberi and malaria, which probably attacked the same patients in the Paraguayan war and believe that this also applies to the malarial polyneuritis cases in the Amazonas province described by many doctors, and which I have never had the chance to see. I cannot deny the existence of the malarial polyneuritis of the North, but I have never met with it. I think that either they are cases of beriberi, so called because the disease attacks a patient in a state of convalescence from malaria, or perhaps malaria and beriberi attack the same patient simultaneously, slight fever being present.

SODRÉ in his magnificent work published in the "Twentieth Century Practice: 1898, Vol. XIV, insists on the value and possibility of beriberic fever, and says that he has observed fever in beriberi to be very irregular and inconstant in its apparance, course and type. According to him, some patients never have fever; in others it exists only when the acute exacerbations of the symptoms follow the first stages of the illness. "The fever may or may not be preceded by shivering; may last only one, or more days, the temperature rising to 39, 5. The febrile state may be continuous, remittent or intermittent. In some cases the fever precedes numbness and other positive signs of the disease, in other cases it follows them. MANSON also mentions fever in beriberi". In an epidemic in Hong-Kong, which he observed, 7 of the beriberi patients had fever after the symptom of numbness, it being more or less accentuated, sometimes very strong and preceded by shivering. SILVA LIMA says," *"Beriberi is an apyretic disease; fever is rarely met with in the course of it and is not a symptom of the illness, but simply a complication, and it seems to me due to the typhoid element."* TORRES HOMEM writes: *"except in the acute form described by* Dr.

SARAIVA, *which I mentioned, I do not think any medical man ever noticed a rise in temperature.*" ALMEIDA COUTO says that he does not consider beriberi a malarial disease, as it was at that time supposed to be, amongst other reasons as it is an apyretic disease. EDWARD B. VEDDER (Beriberi, 1913) says that fever in cases of beriberi is not a characteristic of the disease, and may possibly be caused by a chill, a slight attack of gastro-enteritis or other like complication. MIRANDA AZEVEDO in 1874, basing himself on his own observations and on the opinions of RIBEIRO da CUNHA and COSTA ALVARENGA, declares that beriberi is an essentially apyretic disease.

It has now beem demonstrated that the majority of brazilian observers deny the existence of a special febrile phase in beriberi. This I had the opportunity of confirming even in hyper-acute cases, in contradiction to the observations of SARAIVA in the Paraguyan war and also those of Prof. SODRÉ.

Weakness of the legs. These symptoms of the prodomatic period are followed, sooner or later, by more typical indications of the disease. Weakness of the legs, which at first only shows itself when going upstairs, is followed by the sensation of wheight. When walking the patient feels as if he were traversing a river with a strong current. Then after some days, his legs feel altogether too weak to bear the weight of his body.

Numbness. Numbness or torpor of cutaneous sensibility is a relatively precocious symptom. It begins at the legs, extending symetrically to the feet, and then to the thighs. Thence it reaches to the soles. The patient feels as if he were treading on an india-rubber floor, or on layers of cotton. The numbness now goes upwards and affects the skin of the abdomen. PEKELHARING says that it always spares the groin and the neighbouring parts. SODRÉ speaks of anesthesia round the mouth.

Sometimes, instead of numbness, the patients feel formication or prickliness, so that some complain of a sensation of walking on needles. This perturbation may also affect the mucous membranes. COSTA ALVARENGA describes a very curious and quite unique phenomenon. The patient, when he passed his tongue over the roof of his mouth, felt as if it were covered with threads or hairs.

The painful sensibility gradually disappears. In the beginning transmission of painful feeling is slow; it diminishes gradually until there is complete anaesthesia." SODRÉ also noticed that heat was less perceived. WERNICH describes as a constant phenomenon the existence of an anaesthetic area round the ankles; in this region, slight impressions were not felt. PEKELHARING and WINKLER describe, amongst the earlier symptoms, an increase in the tactile zones, specially in the lower extremities. SODRE, who had the opportunity of verifying and confirming these assertions, claims that this is one of the first symptoms of beriberi. The latter, as well as PEKELHARING and WINKLER, say that in the initial stage of the disease, its presence is revealed by the electrical examination of the muscles and of the nerves. "Faraday"s current shows a diminution of the irritability of the muscles, either direct or indirect, and at the same time a remarkable diminution of the indirect irritability by galvanic currents, as also a somewhat lengthy contraction when anode and cathode are shut.

Locomotory perturbations. In nearly all the forms of beriberi the symptoms follow one after the other and gradually increase. To the initial phenomena, all of which affect the sensibility, succeed others influencing motility.

We not only observed this phenomenon constantly, but also found it described in all the classics.

The feeling of tiredness is followed by more or less accentuated phenomena of paresis.

The course of the disease then assumes its characteristic appearance. The patient finds great difficulty in getting up when lying or sitting down, and only succeeds by reiterated efforts. When beginning to walk, he

stretches his feet wide apart, to have a firmer support; at the same time, he feels bound to keep his attention fixed on the ground, which seems to him, judging from the sensation in the soles of his feet, to be elastic and flexible. He can only keep his equilibrium by the aid of his sight, as the muscles of his legs do not seem to him to obey his will. (SILVA LIMA, MIGUEL COUTO, AZEVEDO SODRÉ).

Later on, the gait becomes even more irregular. The patient lifts his heels very high, implanting the outside border of the foot on the ground. The paralysis of the extensors of the toes causes the point of the foot to sink to the ground, and in order to prevent this, the leg must be lifted very high. FRANCISCO DE CASTRO calls this way of walking "high stepping". Birds suffering from beriberi, also show this characteristic "high stepping". After this simple difficulty in walking, comes an almost complete paralysis beginning in the extensory muscles of the lower extremities, the anterior tibial, the extensory hallucis and the commum extensors of the toes and finally the flexors of the foot. etc. etc.

In the beginning paresis is incomplete but it becomes more and more intense and gradually invades a greater number of muscular groups; after the muscles of the leg, it attacks those of the thigh, then of the fore-arm, the arm, the hand, the abdomen and the thorax. In the upper extremities the extensory muscles are also the first attacked. PEKELHARING observed that the biceps and the great pectoral are the last involved. SODRÉ says that he never saw the sterno-cleido-mastoidian attacked. SILVA LIMA records a case where the paralysis was total and the patient moved only his head.

Complete motor paralysis is very rare. I never saw it. Often, the movements are quite incoordinate. SILVA LIMA says that in his observation VIII «the patient moved his arms and legs, but his movements were limited, incoordinate and jerky. For instance, if he wanted to lift his finger to his mouth or his forehead, he never got it to the right place, nor could he calculate the muscular force necessary for these movements. Abrupt movements of the fingers and hands, due to sudden contraction of the flexors were added to these symptoms on the ninth day.»

Paralysis may also attain other muscles, for example those of the pharynx, as observed by SILVA LIMA and SODRÉ, and also the muscles of the larynx. In experimental beriberi, I observed paralysis of the pharynx and of the oesophagus. Birds in this and at last stage of the disease swallow with the utmost difficulty.

Perturbations of the voice were observed by SILVA LIMA as comparatively frequent phenomena. In his observation VII he noticed a difference in the tone of the voice and has described cases of aphonia. SODRÉ says that paralysis of the larynx may cause sudden death. TORRES HOMEM and MIGUEL COUTO call attention to paralysis of the bladder. In these latter cases miction is slow and requires great efforts on the part of the patients. SILVA LIMA never noticed paralysis of the bladder.

Myalgia. It is constant phenomenon in beriberi be it spontaneous or provoked. SILVA LIMA, as we have already seen, observed that the pain is seated in the paralysed muscles. In the confirmed cases of the disease the patients cannot bear pressure on the muscles of the legs and of the fore-arm. "It is this hyper-sensitiveness which renders walking difficult, when the paralysis of the muscles is not very great". SILVA LIMA also noticed neuralgic pains varying, as to seat, intensity and duration.

It was the neuralgic pains which certainly led to the theory of rheumatism in beriberi, in vogue at the time of JULIO DE MOURA. Pain on pressing of the gastrocnemius is a first-class and well-known diagnostic sign; the same also refers to the muscles of the thigh and the forearm.

Muscular atrophy. In beriberi the muscles are always attacked. Muscular atrophy is recognised by the naked eye when it takes the dry form. This is the rarest form in the north and I seldom met with it. I remember

however one patient who was all skin and bones.

SODRÉ and other Brazilian medical men mention the existence of muscular hypertrophy. Evidently this is an error of interpretation. The muscular hypertrophy could only be apparent, the dominant feature in the pathological course of the disease is atrophy. The illusory hypertrophy is a consequence of oedema which does not attain the muscular fibre, but the intersticial connective tissue. In the first stages there may be a certain degree of hypertrophy of the heart, because it is the perypheric resistance (resulting from the vaso-motor alterations) which causes the modifications of the rhythm and the reduplicating of the sounds. In the motor-muscular system, the atrophic process is however the predominant lesion and as its consequence follows the functional disablement of the respective muscle.

In the oedematous form, the oedema masks everything and the muscle in spite of having its fibres attacked seems hypertrophied, increasing in size because of the diffuse oedema. Hence the mistaken idea of the existence of hypertrophy in beriberi.

Abolition of tendon-reflexes. In the beginning of the disease the tendon-reflexes may appear exaggerated, but the general rule is the abolition of reflexes. The sign of WESTPHAL nearly always exists; the rotular reflex is the first to weaken when the disease sets in, and with the progress of the symptoms it disappears altogether.

Oedema. The perturbations of vaso-motor innervation may be simultaneous with those of the nerves of motility and sensibility, or they may preceed or follow them.

As a rule oedema appears at the same time as numbness and anaesthesia. Sometimes it is evident only after weakness of legs and difficulty in walking have set in. In the galloping form of beriberi, in which the disease takes immediate poesession of the whole of the sympathetic system, oedema, dyspnoea and asystolia predominate.

Oedema nearly always begins on the *crista tibiae*; it is hard and elastic. SODRÉ says that one of the initial symptoms of beriberi is a slight tumefaction of the crest and anterior face. In some regions, oedema, is characteristic of beriberi. In the north of Brazil it is the most general. Dry or atrophetic beriberi, as we have alreadv remarked, is rare. Such is the importance of oedema in the symptomatology of beriberi that SILVA LIMA divides the illness into three clinical forms: paralytic, oedematic and mixed. Here we may say that we do not approve of this division. In the pa ralytic form there is always oedema: in the oedematous form there is always paralysis. Indeed, modern tendency is not inclined to accept these classifications as separations of well-defined types.

VEDDER observes that some patients attacked by rudimentary forms of beriberi, surprise medical men by suddenly, showing cardiac affections, and dying almost suddenly. Cardiac insufficience may occur in all stages of this illness. VEDDER observed in the East, that the disease sometimes remains in the rudimentary stages during months or even years. It is interesting to register that AZEVEDO SODRÉ in 1898, before VEDDER, says: "the symptoms of beriberi may last weeks and months, being sometimes more, sometimes less accentuated; when they are better, the patients feel very well and take up their former occupations. In these cases it sometimes happens that cardiac symptoms suddenly appear; acute and considerable dilatation of the righth cavity of the heart sets in and is almost immediately accompanied by asystolia, and death supervenes sometimes immediately and sometimes after some days.

It is clear that the words of the two doctors coincide. They coincide also with the facts which came under my notice in the north. There is a superposition of symptoms in beriberi, according to the kind of nerves attacked. As long as the great sympathetic and the vagus are not attained the disease may have a comparatively benign course, but at a given moment the cardiac affections intervene, and all cases of beriberi end in the same way, by an attack of acute asystolia.

Oedema in beriberi may attain all the organs. The subcutaneous cellular tissue may assume enormous proportions, as in anasarca. Diffusion in the serous cavities coincides with cutaneous infiltration. A very frequent phenomenon is hydropericardium.

In 256 cases, VEDDER found pericardiac effusion in making autopsies, in the proportion of 66 %. Hydro-thorax and ascites are rarer. The diagnosis of hydro-pericardium is very difficult, it is generally only revealed at the autopsy.

Oedema of the lungs is very rare in the initial stages of beriberi, and is generally one of the final symptoms. Beriberi cough, slight and tiring, accompanied by sanguinolent expectoration, according to some of our classical authors, has nerver come under my notice, even in the most acute forms of the disease. Dyspnoea comes from the weakness of the myocardium because percussion and auscultation do not reveal anything wrong in the lungs, except in the final stages of the disease. PEDRO DE MAGALHÃES attributes the cardiacal affections of beriberi to a constriction in the pulmonary network, caused by the excitation of the thoracical ganglia and of the sympathetic ganglion on which depend the vasomotors of the lungs.

In the acute forms of beriberi I have met with real states of orthopnoea. Breathing is only effected with indescribable anguish. Paralysis of the diaphragm and the muscles of the thorax help to aggravate the situation of the patient. Sitting with his arms as if he were nailed to the bed, with distended neck and cyanotic lips, the sufferer tries to aid his inbreathing with his lips. Profuse cold sweat covers his face on which is imprinted the most anguished image of dispair. SAMPAIO VIANNA counted from 50 to 60 respirations a minute in the acute stage of beriberi.

Beriberi girdle. This is a frequent symptom. The patient feels squeezed, constricted, the sensation beginning in the epigastrium and reaching round the sides. SILVA LIMA describes the girdle as "a feeling of being tied in, with a feeling of fulness and hardness, as if you were weighed upon by a plane or an iron bar". I think that this is because the phrenic nerve is attacked, and thus causes paralysis of the diaphragm. As we are told by SILVA LIMA, TORRES HOMEM and AZEVEDO SODRÉ this sensation may extend to the armpit when the intercostal muscles are paralysed. In the north I found that this makes the prognosis serious. The sensation of the girdle is always accompanied by dyspnoea and precardiac anguish.

Vomiting. Is often found in the beginning stages of the disease and ensues from gastric perturbations. Out of 61 patients, SAMPAIO VIANNA noticed vomiting in 13. In severe cases it is a very bad sign. VEDDER considers it as an indication that the disease is entering on an acute phase. SILVA LIMA tells us that the patients vomit oftener when their liver is very much congested. He is of opinion that the liver is always congested in oedematous cases, as the circulation in the veins is embarassed. In this case the liver is very sensitive to the touch.

According to carefully taken measures by SAMPAIO VIANNA and PEDRO A. DE MAGALHÃES, the size of the liver is always increased (100 %). The spleen is also swollen according to SAMPAIO VIANNA and SCHEUBE.

It is very rare for the sense organs to be affected by beriberi, but Brazilian literature on the disease is full of notes on this subject. SILVA LIMA quotes cases of strabism and diplopia and even a case of blindness which appeared in 24 hours, eight days before the death of the patient.

Cases of amblyopia, diplopia and amaurosis *sine materia* of one eye, while the other one was not affected, were diagnosed and examined opthalmoscopically by HILARIO DE GOUVEA, and observed by MIRANDA AZEVEDO in 1874. Touch seems to be most frequently affected. The patients are even often unable to hold anything in their hands, button their clothes, or even to put on a slipper, unless guided by their sight. (SILVA LIMA).

Convulsions. SILVA LIMA made observations on several patients who had slight convulsions, or choreic movements of the hands and arms. VEDDER thinks that convulsions are rare, but may be provoked by cerebral oedema. In experimental beriberi, however, convulsions are of common occurence as will be seen later on in the account of my experiments. The rareness of convulsions is used as an argument by many scientists to prove that beriberi in man is not an illness of the same nature as that of birds.

The blood. WERNICH found constant hyperglobulia. From the studies of MARTINS COSTA, P. S. MAGALHÃES and WOLFERSTAN THOMAS a general rule may be established, that there is no considerable diminution in the red corpuscles nor increase of white globules in beriberi, especially in the early stages of the disease. As ankylostomiasis is a very common disease in Brazil, some beriberi patients may have been anaemic from the beginning of their illness, the anaemia being considered as proceeding from ankylostomiasis.

The urine does not show anything abnormal in its chemical constitution. SAMPAIO VIANNA found traces of albumen in some of his patients, and in others glucose. Albumen is not found as a rule. The urinary secretion is diminished. In some of the most oedematous cases the quantity of urine passed is very much less, in some cases only 200 to 300 grammes and even as little as 75 grammes in 24 hours.

The urine may stop altogether. FRANCISCO DE CASTRO was the first to call attention to beriberi uremia, without albuminuria. SAMPAIO VIANNA met two patients with frank symptoms of uremia. The diminution of the urinary secretion is due to vaso-motor *oedema* of the cortical part of the kidney, and as the tissues of this part are inextensible, the pressure applied to the afferent vessels explains everything (NINA RODRIGUES). The density of the urine always increases.

Pulse. The pulse is characterized by its frequency, irregularity and lowered tension. In the acute cases it attains 150 a minute, and sometimes is to rapid to be counted.

Heart. The examination of the heart is of the greatest interest, as the alterations observed are almost sufficient to distinguish this polyneuritis from all others. In the beginning of the disease palpation indicates the increase of cardiacal impulse, naturally due to the resistance in the network of pulmonary circulation. In the later stages, when the heart is dilated, the cardiacal impulse is of counse always dimished.

Percussion always gives an increase of precardiac dullness especially on the right side, which is increassed in size and reveals a larger zone of dullness of 2 or 3 centimeters to the right and deviation of the point to the left (a few centimeters). This increased dullness was observed by SAMPAIO VIANNA in 70 o/o of his patients, and is not only due to the swelling of the organ, by dilatation or hypertrophy, but may also be attributed to hydro-pericardium, always met with in the final stages of the disease. The dilatation, is in the beginning confined to the right ventricle, then it spreads over all the right half of the heart. SAMPAIO VIANNA says that a very certain sign of beriberi cardiopathy is the coinciding of epigastric pulsation with the absence of precardiac shock which is an immediate consequence of the dilatation of the right side of the heart. Unfortunately, this modification of the shock of the apex is not absolutely constant; in the majority of cases it is only weakened, as is ascertained on touch. SAMPAIO VIANNA affirms that in all the patients observed in the hospital at Copacabana, he found the diastolic pulsation was greatly diminished or almost absent

Palpitations. These constitute a symptom from the very beginning; at first, they are brought on by the least exercise or effort, even by a moral impression. Later on, tachycardia is a permanent phenomenon. The acceleration is accompanied by palpitations and is, according to PEKELHARING and WINKLER, simultaneous with the first manifestations of the disease. The slowing down

of the rhythm is rarer; I have never met with it. SCHEUBE often observed it, especially some hours before death. The palpitations also cause the precardiacal pain, which in slight cases is only a feeling of oppression, though in very acute cases it assumes the proportions of *angina pectoris*.

FRANCISCO DE CASTRO was the first to describe the beriberi *angina pectoris* «with excruciating retro-sternal pains, which extend to the neck, back of the neck and left arm». PEKELHARING and WINKLER describe similar observations. In the cases that came under my notice, I saw that the pains were far more excruciating than those of simple angina pectoris. The attendant dyspnoea, pains, the livid countenance contrasting with the cyanotic lips, the groans, the anxiety, the wild look of the patient together form a picture of as dramatic and pungent intensity, as the worst attack of angina pectoris. Under such conditions of well-defined asystolya it is impossible to obtain any clear account of his feelings from the half-unconscious patient.

Ausculation reveals other most important signs of heart affection. One cannot deny that SILVA LIMA deserves all the honour of the discovery of the predominance and gravity of the cardiac phenomenona in beriberi. He calls attention to the perturbations of rhythm: *"a complete disorder in the succession and frequence of the systolic and diastolic movements, which render it impossible to count the cardiac revolutions and the pulse"*. He also called attention to the reduplicating of the second sound, "thus giving rise to three distinct sounds; the first is the ventricular systole and after a short interval, in comes the diastole sound which is re duplicated and followed by a long pause". Reduplication of the first sound, although mentioned by SILVA LIMA, TORRES HOMEM and FRANCISCO DE CASTRO, is almost denied by PEDRO DE ALMEIDA MAGALHÃES and SAMPAIO VIANNA. The reduplication of the second sound has been met with and well described by all the Brazilian medical men who studied beriberi cardiopathy, from SILVA LIMA down to the present day. AZEVEDO SODRÉ and PEKELHARING say the point of the heart is the best place for hearing the doubled sound; ALMEIDA MAGALHÃES and SAMPAIO think that the best point for auscultation of the second sound is the focus of the pulmonary artery.

There is no doubt that these latter observers are right. MAGALHÃES says that of the two sounds, the aortic precedes the pulmonary. SODRÉ denies this invariability. However all brazilian pathologists agree that there is accentuation and reinforcement of the pulmonary sound, a fact to which FRANCISCO DE CASTRO called attention for the first time, and which was confirmed by SCHEUBE, PEKELHARING and WINKLER, AZEVEDO SODRÉ, PEDRO DE MAGALHÃES and SAMPAIO VIANNA.

Galloping sound. This sound was first mentioned by PEDRO ALMEIDA DE MAGALHÃES, SAMPAIO VIANNA noticed that the adiventitious sound, whose addition to the normal sounds, causes the sound, of galloping is localised in the epigastric region and is sometimes more distinctly heard at a certain distance from the left margin of the sternum (margin of the right ventricle).

Murmurs. The murmurs are according to P. A. MAGALHÃES and SAMPAIO VIANNA generally mezzo-systolic, rarely systolic. "These sounds are soft, superficial, variable and not lasting." SAMPAIO VIANNA heard them distinctly in 24 patients out of 24.

Pathogeny of the cardiac alterations. SCHEUBE says they ensue from the alteration of the vagus. AZEVEDO SODRÉ and LACERDA attribute then to the alterations of the sympathetic system. NINA RODRIGUES, without denying the influence of the alterations of the vagi, is of opinion that the lesion of the sympathetic is the chief factor in the cardiopathy observed in beriberi.

PEDRO DE MAGALHÃES thinks that the cardiac alterations ensue from the vasomotor disorders in the pulmonary circulation "the constriction of the pulmonary network

of vessels causes an increase of pressure on the pulmonary artery".

"The pulmonary circulation having its tension increased, the pulmonary sound becomes accentuated, the ventricle is dilated, and byreathing, on the increased it pressure becomes hypertrophied. The right ventricle being dilated, the fibres of the myocardium lose their tenacity; hence the galloping sound. The right ventricular cavity becomes dilated, so that the diastolic aspiration is lessened, and the pulmonary valve, closing after the aortic, causes the doubling of the second sound". This explanation is reasonable and logical. MAGALHÃES is of opinion that tachycardia is provoked by the irritation of the sympathetic system and the degeneration of the vagus.

We avoid dividing beriberi into different forms as one cannot separate well-defined varities. Sometimes beriberi progresses slowly and becomes chronic; at other times it progresses swiftly and becomes very acute, with the intervention of the serious phenomena above-described. Often however the evolution of the disease takes place in a few hours or days; it is then known as galloping. Beriberi nearly always terminates in asystolia, either by direct attack or by the degeneration of the nerves innervating the heart. The degeyeration of the vagi and the lesions of the great sympathetic explain the evolution of the symptoms which have been noticed by many pathologists.

VEDDER does not accept the classification of clinical forms, but establishes a list of three kinds of principal symptoms: 1st) symptoms of peripheric neuritis 2d) cardiac insufficiency 3d) general tendency to oedema.

On comparing Brazilian studies with those of the Eastern authors I came to the conclusion that there is no difference between brazilian beriberi and they Japanese, Malayan or Philippine kind. No description could fit the galloping forms of the Amazonas better than that of the most acute forms seen in Japan by SCHEUBE. There is no difference between some of the chronic, torpid, attenuated forms observed by me in the Amazonas province, and the Segel-schiff beriberi of NOCHT. The experimental cases in men, recently described by STRONG and CROWELL, might be pictures of those described by SILVA LIMA and SODRÉ.

We therefore conclude that the beriberi in Brazil is the same as Eastern beriberi as SILVA LIMA recognised.

Experimental beriberi.

In 1890 EIJKMAN noticed for the first time, that the birds fed with polished and cooked rice in his laboratory in Batavia (Java), showed ataxic symptoms, paralysis and convulsions after some time; in short, they revealed symptoms of an illness resembling human beriberi. His communication was received either with absolute indifference or, with complete unbelief. He did not however allow himself to be discouraged, but went on with his studies. At first he thought that *polynevritis gallinarum*, as the new disease was called, was a toxic polyneuritis caused by a substance that poisoned the nerves and resulted from fermentation or change in the rice when shelled, and which harmed birds when it remained in their crop. Since the birds showed no signs of illness when they were fed on unshelled rice, he thought that the pericarp protected the seed from the germs of fermentation.

In another series of investigations EIJKMAN renounced this hypothesis of intoxication as he saw that the shell of the rice contained a matter essential to the nourishment of birds and that without it they had polyneuritis. This substance is found in the red pellicule which encloses the seed. The pigeons nourished with unpolished rice with the pellicule, did not get polyneuritis even when the rice was cooked in the ordinary way. Consequently he realised that the illness was caused by a defficient nourishment. EIJKMAN therefore came to the conclusion that in the pellicule of rice there is a substance of such nutrive and protective value to the nerves that when it is wanting they became disintegrated because theyr structure is attacked."

Another great advance was made when EIJKMAN verified the fact that excessive heat destroys the protective action of the rice-pellicule as well as that of all other cereals. He fed birds on unshelled, rice, heated to 120° C. for two hours, and saw that they died of polyneuritis. Oats, barley and millet eaten raw prevent polyneuritis. Cooked in the ordinary manner these cereals do not lose their protective action, but if they are kept two hours in the autoclave at 120°, they lose it and provoke polyneuritis EIJKMAN therefore arrived at the conclusion that cereals contain a substance of special nutritive quality with a neuro-protective power, which is lost when the cereals are submitted to high temperatures.

These observations were elaborated some years afterwards by SCHAUMANN, who proved that long storage in the hold of hot and damps ships, or in laboratories in glass jars destroyed this protective substance. It was proved also that the birds attacked by polyneuritis were cured, if fed on meal of rice-husks, or if their insufficient alimentation was supplemented by other foods of greater protective value such as oats, barley, millet, etc.

No sooner was the existence of a highly nutitrive and protective substance in the rice-husks known, than scientists in all parts of the world began to investigate the nature of it. As the rice-husks are rich in phytin, EIJKMAN went on to prove that phyin does not cure, nor does it prevent the illness, but that the substance sought for, is present in the watery extract of rice-meal after phytin was extracted.

These observations were confirmed by FRAZER and STANTON, also by CHAMBERLAIN and VEDDER and were the basis of the method used in extracting the *Protective substance* from the remains left after cleaning rice. A substance rich in phosphate was also extracted from rice-flour; it did not however protect the birds to which it was given by EIJKMAN in their polished rice food.»

Carrying his observations still further, EIJKMAN came to the following conclusions. "An apparently physiological diet may provoke the disease and even lead to death" and "although *polynevritis gallinarum* is not in all points identical with beriberi in men, it is nevertheless caused by the same conditions. Human beriberi can be prevented and cured by the same diet as that which protects and cures birds.

In his thesis ARLINDO DE ASSIS, a pupil of FRAGA, proposes that the *polyneuritis gallinarum* be called EIJKMAN'S disease in homage to the important services rendered to science by the Dutch professor. I do not follow his example, but am of opinion that the name of beriberi ought to be kept, even if it be insufficient to express all that is meant by polyneuritis, brought on by insufficient alimentation. This I think due to SILVA LIMA, who chose this one, from amongst all other names for the same disease in use at the time in which the disease was observed in our country, and which has remained in scientific terminology. I am also convinced that there is not such a wide difference between experimental beriberi (polyneuritis aviarum) and human beriberi and I think we should adopt a generic denomination for both.

GRIJNS demonstrated that sago, tapioca and meat, heated in an autoclave to 120°, provoked polyneuritis, almost as easily as polished rice. In 1901 he found that beans of the kind called *Katjangidjo* have a protective and curative action on the birds fed on polished rice. He however adopted the opinion that the disease was a real poisoning which could be neutralised and even prevented by feeding on rice-husks or Kat jangidjo. GRIJNS however discarded the idea of infection.

In 1910 SCHAUMAN, in a series of experiments on *polyneuritis gallinarum*, came to the conclusion that it is a disease of metabolism, provoked by the absence of certain not determined organic combinations of phosphates in the food. He even determined that good rice ought to contain 0,4 penta-

oxyde of phosphorus (P_2O_5). SCHAUMANN believed that these organic combinations of phosphorus would be multiple and diverse: phosphatides, nucleic acid and probably many other substances which would act as stimulants, provoking and stimulating organic changes; they would be activating substances.

However, in 1911 FUNK isolated a substance from rice-husks which had a specific curative power on the experimental beriberi of birds. This discovery was a landmark in the study of the question. By means of phosphoro-tungstenic acid and also by means of nitrate of silver in the presence of baryte, he managed to precipitate an active substance of great curative value from the extract of rice-husks. Combining the two methods, he obtained an organic crystalline basic substance which melted at 233°. C. FUNK gave the chemical formula ($C_{17}H_{20}N_2O_7$) to this basis. This substance was obtained from yeast and from many other alimentary substances. The small dose of 2 centigr. was sufficient to cure pigeons attacked by aviarian beriberi. FUNK thought that the substance thus isolated "was of complex structure and analogous to amides and because of its great biological functions named it VITAMINE, as this vital amide is one of those nitrogenic substances, infinitesimal quantities of which are indispensable in food, for the integrity of the nervous system of birds, men and other animals. "These studies were confirmed in Manilla, by VEDDER and WILLIAMS, who, by the same methods, isolated the same crystallisable substance, which was effective in curing a pigeon attacked by polyneuritis, in a dose of only 30 milligrs.

In March, 1912, EVANS, SIMPSON and WEBSTER, managed to isolate from yeast an organic basis which they called TURULINA and which also cured experimental beriberi, and for which they gave the formula: ($C_7H_{17}N_2O_5$).

In 1912 TZUSUKI extracted from rice meal a substance which he called antiberiberina. This was only an alcoholic extract identical with that which CHAMBERLAIN and VEDDER used for curing the disease in men. *Orizanina* found by SIMARUM and ODAKO (July 1912), obtained from the alcoholic extract of rice-meal by means of tannic acid, also cured beriberi in birds.

FUNK'S discovery gave rise to an infinity of interesting studies and although chemists are not all agreed about the exact formula and the chemical constitution, the name vitamine ought to be kept for the substance, which will doubtlessly latter on be found by means of more perfect analysis. The term VITAMINE ought even to become a generic name, not only for the substances found in rice-husks, yeast, Katjangidjo and other foodstuffs such as ox-brains, milk, horseflesh, the testicles of bulls etc. which have a protective action against beriberi, but also for the substances in food which act against scurvy.

Birds are not the only animals who show symptoms like those of beriberi in men. SCHAUMANN, in 1910, was successful in provoking polyneuritis in guinea-pigs, rats goats and a monkey. A dog, fed on meat sterilised at 120°, became completely paralysed. A goat fed on polihsed rice, became paralytic 3 or 4 months afterwards. HULSHOFF POL demonstrated the disease studied by SCHAUMANN in these animals to be the same as polyneuritis of birds and human beriberi.

SHIGA and KUSANA, in spite of their being at first opposed to the identfication of human beriberi with the experimental beriberi of SCHAUMANN, made some interesting experiments on monkeys.

The first monkey fed on cooked rice died of tuberculosis at the end of a few weeks. The second, having lost in weight, showed paralysis of the legs at the end of 37 days. At first the turn the disease would take was uncertain, but afterwards the paralysis progressed and became complete. Anaesthesia of the legs was present. The reflexes were at first augmented and ultimately abolished. Ausculation showed accentuation of the aortic sound and the pulmonary sound. The slightest exercise brought on palpations and there was considerable dys-

pnoea. In the last days oedema of the legs set in. At the end, cardiac troubles increased. Cardiac energy diminished. The pulse was weak and soft. The temperature went down, and death took place, 10 days after the illness declared itself. (Apud VEDDER).

We see that the monkey had all the symptoms of human beriberi: paralysis of the legs, gradually ascending, anaesthesia, loss of reflexes, oedema and cardiac symptoms. In other observations these scientists found the same typical lesions of beriberi and hypertrophy of the heart.

KUSANA and SHIGA drew the following conclusions from their studies: "It is a recognised fact that the dilatation and hypertrophy, essentially of the right side of the heart, are very characteristic of beriberi, so that this disease is very easily distinguished from polyneuritic intoxications. The illness of the monkeys was the same as human beriberi".

TSUSUKI also provoked experimental beriberi in dogs, cats, guinea-pigs, rabbits, rats and monkeys by feeding them on polished rice. ANDREWS (1912) also provoked the disease in newborn dogs. He had noticed that the disease "Taon" which causes great mortality amongst babies before they are weaned, is nothing else but infantile beriberi and that "Taon", was caused by the poor milk of the mothers who lived on polished rice. He put some little dogs to the breasts of the mothers whose chidren had died. The puppies were nourished thus for one month, at the end of which time they began to show incoordination of their movements, weakness of the extremities, especially of the hiny legs, in many cases followed by paralysis. All had oedema of the subcutaneous cellular tissue. Marchi's method, showed degeneration of the peripheric nerves in all of them.

In 1906 WEILL and MOURIQUAND published an important work on experimental and clinical diseases caused by insufficient food in the "Revue de Médecine". This work, as well as the counter-proof, was made in the Lyons Laboratory. WEILL and MOURIQUAND called the diseases caused by the want of vitamines, diseases of want. These diseases were provoked experimentally or observed clinically and came from want of a certain ferment of which the smallest dose is necessary for normal nutrition. This new denomination was adopted by ARLINDO DE ASSIS, pupil of FRAGA, in his thesis in 1918. The english writers had already given the name of defficiency diseases to this group of illnesses. FUNK had proposed the name fo AVITAMINOSES which I find euphonic and expressive.

WEILL and MOURIQUAND'S work was another brilliant confirmation of that of EIJKMAN, FRAZER and STANTON, SUZUKI, SHIMAMURA, SCHAUMANN, FUNK, VEDDER, STRONG and all the others. He reproduced beriberi in birds and studied the symptomatology of the disease practically and systematically. He also produced scurvy in rabbits and polyneuritis in cats by destroying the vitamines of their food by long sterilisation in the autoclave. It was he who invented the expression that vitamines are living substances which can be destroyed by long sterilisation, saying that: "Life is necessary to life". WEILL and MOURIQRAND concluded that aviarian polyneuritis and human beriberi are caused by alimentary insufficiency. Though there are some differences in certain manifestations, one cannot expect the same reactions in men and animals in all points. Looking through all this enormous amount of literature, we notice that this highly interesting experimental beriberi did not awaken much enthusiasm amongst Brazilian authors.

The first brazilian work on experimental beriberi was published by ARLINDO de ASSIS. Following the method of WEILL and MOURIQUAND, he made a series of most interesting experiments on defficiency. His work deserves to he read by all who are interested in such. As hommage to EIJKMAN, he proposed to call experimental beriberi EIJKMAN'S disease. I have already explained my reasons for differing from him. As these studies are of great national interest thy ought

to be written in simple language, so that the general public may be made cognisant of them. Thus the expression experimental beriberi is more suitable and shows the connection between it and human beriberi, allowing the reader to draw the right kind of conclusions and to learn what is of practical use to him.

In August of this year, (1919) I resolved to make a series of experiments on experimental beriberi with the cereals that are used for food in the north and the interior of Brazil. Dr. CARLOS CHAGAS was kind enough to allow them to be carried out in the aviary at Manguinhos. The library and the laboratories were also placed at my disposal.

The cereals chosen for the experiments were polished rice, fresh indian corn sterilised (1) by indian corn sulphu rate of carbon; fresh and first class surui flour; mouldy Surui flour kept stored for a long time; farinha d'agua (mandioc flour specially prepared), polished rice sterilised in the autoclave at 120° during an hour; Surui flour of the best quality sterilised in the autoclave at 120° during an hour; first class maize-flour sterilised in the same way.

The object of these experiments was first to get to know *polynevritis gallinarum* de visu, by provoking the disease in fowls and pigeons and then to verify the analogy or the differences between this disease and beriberi in men: secondly) to ascertain the quantity of vitamines in Surui flour and in mandioc meal and whether the exclusive use these substances causes *polynevritis gallinarum* quicker or more slowly than polished rice; thirdly) to find out if the sterilization processes in use in Rio de Janeiro be hurtful to the health; fourthly) whether lengthened sterilisation of nutritive substances or their getting old by long storage causes the destruction of vitamines thus provoking beriberi.

(1) Indian Corn was supplied by the Empreza Este de Cereaes.

In spite of the fact that the work of ARLINDO DE ASSIS is worthy of all confidence, new contribution on the subject may throw new light on it, especially on those of its aspects which are not absolutely clear; it may also resolve some unsolved problems concerning the diseases caused by defficiency. It is enough to mention all there is still to be investigated as to the nutritive value of the milk used in Rio de Janeiro and the sterilized milk used de larga maxima in The Amazonas province.

The high infant mortality amongst us must be ascribed as in a great part due to the bad quality of food, to the milk deprived of its nutritive qualities by being over frozen or hypersterilised, to the flour of doubtful nutritive value, especially of that which comes from foreign countries and has been kept for an unknown lenghth of time.

I began my observations on the 15th of August 1918, chose carefully examined, healthy animals, and had their compartments rigorously desinfected.

Those on which the experiments were to be made came from the same place as the witnesses, which were plentifully supplied with Indian corn.

The first group were fowls fed on Amazonas flour (farinha d'agua), of good quality, sterilised at 120°.

The fowls resisted from the 15th. of August until the 21st of October, without manifesting any symptoms of *polyneuritis*. They lost weight, and lost appetites but showed no signs of beriberi.

My technique was modelled on that of WEILL and MOURIQUAND. The animals were weighed every day and their food was changed and measured also every day. One of the fowls was artificially, fed as it would not eat, but it died of asphyxia during the process.

The conclusions drawn by me from these experiments do not agree with those of WALCOTT and ARLINDO DE ASSIS.

WALCOTT obtained *polyneuritis* by using Amazonas flour, but his experiments lasted

much longer than ours and he does not tell us how they were made. ARLINDO DE ASSIS, who modelled his experiments on those of MOURIQUAND, obtained *polyneuritis* with the mandioc flour commonly used in Brazil, but did not make experiments with Amazonas flour.

Amazonas flour must be subject to a process of preparation which deprives it of its nutritive value which seems to be less than that of white flour used in Brazil. However it may be, my experimental animals, which were fed on nothing else, did not that show any symptoms of *polyneuritis.*

We might therefore conclude that Amazonas flour is not a defficient food; when fresh and good, is even better than polished rice. As my experiments were not long or complete, other experiments should be made the same direction.

Another group of fowls (3) was fed on old white flour. Two succumbed with all tne symptoms of *polyneuritis*, the third resisted all the time the experiment lasted onfv manifesting loss of weight and want of appetite. We here give the details of the observations made on one of the fowls.

"Cock. Nº. 4 Weight: 1,880 gr. On the 16th it got a ration of 120 gr. of white flour of the Surui kind, of very bad quality; it ate 75 grs. of the ration in 24 hours.

During the first days it ate well, averaging 60 to 80 grs. of flour a day. From the 24th. to the 30th., in spite of not showing any outward symptoms of disease, it lost weight and appetite.

It continued to lose weight, being only 1,800 grs. on the 26th and eating only 20 grs of its ration a day. On the 30th. it weighd only 1,660 grs. From the 30th. on, it would hardly eat, taking only about 3 or 4 grs. of flour a day and on some days would not even take any. On the 5th. it weighed only 1,490 grs. it was sad its wings drooping while it lay down and, seemed apathetic.

On the 6th. and 7th. it could hardly walk, showing the characteristic beriberi stepping. On the 8th. it weighed 1,290 grs. The wings and legs were paralysed. When strongly excited, it made incoordinated movement of the feet and legs. On the 9th. the paralysis was complete, the movements convulsive and tetaniform: the weight fell to 1185 grs. and the fowl died.

A third group was fed on national polished rice, of the kind here called needle rice, of the best quality, sterilised at 150º. This group was composed of pigeons.

Afer some days the classic symptoms of *polynevritis* declared themselves.

Pigeon n. 8, shown in photograph n. 1, became paralysed after a few days.

Pigeon, n. 9, (fig. n. 2) died after being fed on polished and sterilised rice for 20 days; all the clasic symptoms were present.

Heating in an autoclave destroys the vitamines and increases the already existant deficiency.

These observations only serve to corroborate those already made and published.

We still wanted to know whether the immunisation of cereals as done here, is harmful by destroying the vitamine.s

For this purpose, a fourth group of pigeons was fed exclusively on Indian corn purveyed as sterilised, by the Companhia Esterelisadora de Cereaes. Their sterilisation is effected by means of sulphurate of carbon steam. The Indian corn keeps its natural appearance and its germinative power.

The feeding of these pigeons went on for 2 and 1/2 months; they all increased in weight, had good appetites in spite of the monotony of their food, and none showed any symptoms of *polynevritis gallinarum.*

As pigeons resisted the diet of polished rice better than gallinaceous birds, I rosolved to make experiments with the latter, feeding them exclusively on polished rice, raw or heated in the autoclave at 120º.

I herewith give the description of a cock as type observation. On the 29th. of August, I began the experiment. The weight of the bird was 1260 grs. and its food was national polished "needle" rice given in rations of 120 grs. I must here sya that national rice

is not so smooth as that which comes from foreign parts, there being always a vestige of the red shell in each grain.

The weight and the alimentation were as follows:

On the 21st.	of August	— weight:	1.260 grs.	ate	80 grs.
22nd.	«	— «	1.250	«	«	55 «
23rd.	«	— «	1.220	«	«	55
24th.	«	— «	1.230	«	«	100
25th.	«	— «	1.225	«	«	40
26th.	«	— «	1.195	«	«	60 «
27th.	«	— «	1.220	«	«	80
28th.	«	— «	1.170	«	«	40
29th.		-	1.175			40
30th.	«	— «	1.160	«	«	40
31th.	«	— «	1.160	«	«	60
1st.	of September	— «	1.140	«	«	50 «
2nd.	«	— «	1.140	«	«	40 «
3rd.	«	— «	1.115	«	«	35
4th.	«	— «	1.080	«	«	20
5th,	«	— «	1.055	«	«	5th. «
6th.	«	— «	1.040	«	«	0
7th.	«	— «	1.020	«	«	0

The bird found great difficulty in flying and seemed very sad.

On the 8th. of September the weight was 1999 grs. It ate 5 grs. and was still sad. It could not fly, when greatly excited, but ran. It tried to lie down continuously showing great weakness in the legs, as is seen in fig. 3. It stepped like people with beriberi.

On the 19th. of September the weight was 980 grs. It ate nothing and kept lying down, walking with great difficulty as can be seen (fig. 4,) opening its legs very wide to walk.

On the 10th. of September the weight was 965 grs. it ate nothing but was fed by *gavage* on 18 grammes of rice to avoid inanition.

On 11 th.	of September-	weight	955 grs.	gavage	40 grs.
12 th.	«	«	975		40
13 th.	«	«	985		40

The ataxy phenomena were accentuated. The food stayed in the crop without being digested.

On the 14th. of September the weight was 995 grs. No *gavage* was made; the crop was still full of food.

On the 15th. of September weight 995 grs. No *gavage*. Crop full.

On the 16th. of September weight 990 grs. *Gavage* 40 grs. Increase of paralysis. Kept lying down.

On the 17th. September weight 980 grs. *Gavage* 40 grs.

On the 16th. of September weight 980 grs. No gavage. Crop full. Food not digested.

On the 19th. of September the weight was 980 grs. No *gavage*. The extensors were completely paralysed, the feet permanently bent. Crop quite full of food administered before.

On the 20th. of September the weight was 980 grs. No *gavage*.

On 21st. September the weight was 960 grs. No *gavage*. Crop still full of food. Total paralysis. Cyanotic crest. Slight convulsions of the wing-muscles. Death.

Fig. 5 the photograph of a hen dieted with polished raw rice. (Hen n. 15).

From this we deduce that the birds eat raw rice for some days with a good or even exaggerated appetite. From the 20th. day on, the want of appetite increases, becoming quite absolute between the twentieth and thirtieth days when the morbid phenomena become accentuated.

Fowls get *polyneuritis* more quickly than pigeons when fed on polished rice sterilised at 120° for an hour. Their want of appetite is almost absolute in the first 19 days. The loss of weight is also considerable. The beriberi symptoms appear on the 15th. day, paralysis on the 17th and death on the 19th. Death may be said to ensue from inanition, for the fowls refuse to eat.

As the fowls seemed to find rice disagreeable, we resolved to feed them on Indian corn which they seem to like better, and which, though sterilised, does not lose its organoleptic qualities.

A group of fowls was dieted on Indian corn sterilised in the autoclave at 120°: The morbid phenomena of beriberi appeared after the 20th. day, the want of appetite ap-

pearing much later than is the case with sterilized rice flour.

Figs. 7, 8 show the different phases of *polyneuritis* in cock nº. 13, dieted with sterilised Indian corn. The experiment began on the 22nd. of August, the fowl dying on the 27th. of September.

On the 23rd. day of treatment there was great weakness of the legs; the fowl lay down on its thighs all day; it stepped high and could run only with a great effort. Complete want of appetite. To avoid death from inanition, it was fed by *gavage*. On the 24th. it could not walk (as shown in fig. n. 8.)

The three last days before death there was complete paralysis, and only when strongly excited could it move or contract its legs and wings.

Another group was dieted on the finest mandioc flour. The results confirmed the observations of ARLINDO DE ASSIS.

I thus proved that a diet of fine and good mandioc flour is less defficient than a diet of polished rice. Amazonas flour is less defficient, and is less likely to induce *polyneuritis gallinarum* than white flour of the Surui kind.

Polyneuritis aviarum and beriberi have so many symptoms in common that we have no hesitation in joining those who accept their etiological and symptomatic identity.

Our experiments though incomplete, corroborate this conviction as well as the authority of those of SHIGA, SCHAUMANN, WEILL and MOURIQUAND.

Etiology of beriberi.

Since the time of SILVA LIMA until now, the idea that beriberi is an infections disease was prevalent amongst our best-known Brazilian doctors. Even SILVA LIMA, though we find passages in his works which support the alimentation theory. was obliged by the force of circunstances to admit the hypothesis of infection. This hypothesis was adopted by all the well-known Brazilian doctors who studied beriberi; leaving some aside, we only call to mind the names of TORRES HOMEM, MARTINS COSTA, SARAIVA, FRANCISCO DE CASTRO, SODRÉ, LACERDA, FAJARDO, PEDRO ALMEIDA MAGALHÃES, SAMPAIO VIANNA and AUSTREGESILO.

FAJARDO and LACERDA even discovered pathogenic micro-organisms although latterly, LACERDA came to accopt the ideas which came from the East.

MIGUEL COUTO, repudiated the alimentary theory as the cause of our beriberi; he believes that the etiology of Brazilian beriberi is as yet unknown. Amongst those who belived in the alimentary theory we must count SILVA LIMA, in whose book we find proofs against the transmission of the disease and who speaking of its propagation says: "Not to presume to affirm anything certain about the diffusion of the disease, that is without geting away from facts, I must say the disease does not seem to propagate itself by contagion or infection, but to depend on a widely spread morbid cause and unknown serious hygienic conditions".

For a long time it was thought that beriberi depended on malaria. ALMEIDA COUTO in his competition thesis in 1871, says that he does not consider beriberi as a malarial disease on account of its being apyretic. He points out as causes, inferior food, alcoholism. SILVA LIMA, also repudiating the idea of malaria, wisely observes: beriberi epidemics have always been very frequent and fatal in unhealthy places, but for causes which have nothing to do with malaria, and particularly on board high seas, ships, in urbane prisons, penitentiaries, etc.

TORRES HOMEM recognizes that "insufficient nourishment both in quantity and quality is an important factor in provoking the appearence of beriberi, which more frequently attacks persons who are convalescent and have a great struggle for life."

MIRANDA AZEVEDO in 1874, was courageous enough to attack all the old theories in his inaugural thesis in which the defends the insufficient nourishment theory. At that time, the malarial etiology of beriberi was supported by the best-known medical

men of the country such as TORRES HOMEM MACEDO SOARES, A. FARIA, SARAIVA and others. On this occasion, LE ROY DE MÉRICOURT thus expressed his opinion: Beriberi breaks out amongst the crews and passengers of ships after long long journeys, amongst soldiers after a campaign, miners, prisoners, orphans (of course he is refering to homes) especiatly amongst the poorer classes, wno have been living on a meagre and uniform diet, which either from circumstances or for the sake of economy is insufficient for their needs.

MIRANDA AZEVEDO was struck by this passage and the allusions to the forms observed in Paraguay, and encouraged by his own observations, was not afraid to criticize the most celebrated medical men of the day in his thesis and affirmed that insufficient mourishment is the cause of beriberi. He says:—"I understand by insufficient nourishment, not only the lack in quantity of food necessary to keep one in health, but also the want of variety of food, a pernicious uniformity, for varied nourishment is imperiously called for to sustain the organism. He afterwards says "Reading and study of the different hypotheses as to the origin of beriberi induced me to pay attention to popular beliefs and to think that rice is the cause of beriberi in Brazil and in India.

The only calss of diseases in which this strange one can be classified, is that of the diseases called, by JACCOUD and other modern pathologists, constitutional distrophy on account of the etiology and the special symptoms.

JAYME SILVADO wrote some very interesting memoirs of Brazil in 1907: "*Beriberi in the Navy of Brazil*". In spite of his being influenced by the theory of infection he gave some good advice to the Superior Administration of the Navy, which, had it been taken, would have hastened the disappearance of the disease amongst our sailors. Referring to the question of rations he speaks of the sailors throwing their bread into the sea either because it was bad or because they did not like it. He also says: As to the food, our rations are not what they ought to be neither as to quantity nor as to the way of cooking the food. The sailors do not get good food as the purveyors want to make as much profit as they can and purvey bad food for the price of good. With great discernment JAYME SILVADO, in his monograph, calls attention to the monotony of the food and the great want of variety in same.

Speaking of the Indian corn meal given on board ship (1907) three times a week, as obrigatory food, he says: "Only people who are used to Indian corn meal can like it, and the kind sent on board ship is enough to make anyone sick of it. It is a already sour when provided, and gets even more sour in the hot damp holds of the ship, becoming absolutely uneatable."

In this work, Dr. SILVADO draws the attention of the government to the food in the Japanese Navy, where by the advice of TAKAKI, barley-meal was added to the rations, so as to counteract the bad effects of rice. SILVADO refers to the communication read at the Congress in Paris in 1900 by Baron SANEIOSKI, and though he did not believe all that was therein, and did not believe beriberi was caused by eating rice, he avows that bad rice brings on polyneuritis.

He shows however a tendency towards adopting the nourishment theory, by giving the statistics which show the difference in the Jaanese Navy before and after the adoption of TAKAKI'S theory.

Statistics of the decrease of beriberi beginning in 1884, when the changes were made in the rations of the Japanese Navy.

BERIBERI.

Years	1880	1881	1882	1883	1884	1885	1886	1887	1888	1889
Cases	1725	1163	1929	1236	718	41	3	0	0	3

As we see from these statistics, if the necessary attention had been given to them,

the greatest practical results would have been obtained, and beriberi would already be no more in our Navy.

In 1914, Professor AUSTREGESILO published a most important monograph on an epidemic of beriberi in the National Lunatic Asylum, which he called scorbutic polyneuritis, in the REVUE de NEVROLOGIE. Analysed by the light of the new doctrines, this work enlightens the etiology of the other epidemics of beriberi which have been noticed there. Studying all his magnificent observations in detail, we see that beriberi and scurvy broke out at the same time, and were most likely both caused by defficient alimentation! The polyneuritis described by Professor AUSTREGESILO had all the characteristic symptoms of beriberi.

Let us take for instance one of his observations: "D. Igl. I do not know when the illness began, as the patient is insane but on the 24th. of February he was shown to me, had attacks of dyspnoea tachycardia, difficulty in walking, oedema of the lower limbs; he looked like a very weak person, with ptyriasis versicolor on his neck, acne on the back, petechias on the lower limbs, scorbutic signs, echimoses in the popliteal space and also purpura spots on his legs; the general oedema was more accentuated in the lower limbs The patient stepped high; the rotular reflexes were exaggerated aud lasted a fortnight. Afterwards the reflexes diminished without disappearing. He had anaesthesia in his lower limbs, aud hyperaesthesia of the trunk. Examination of the faeces revealed eggs of ankylostomum. There was reduplication of the first and second sounds of the heart. The murmur of the pulmonary artery vibrated more than in health.

This patient was put in the infirmary of the Asylum, but his family removed him so as to nurse him. He then escaped from the vigilance of the family after he was cured of the intercurrent disease.

We see that this was a classic form of beriberi, where there were symptoms of scurvy caused by of defficient food, which were almost cured by a change of diet.

2nd. Observation. L. M. years old, Brazilian etc. 16th. of May 1916. During the slight epidemic of scurvy this year, he had classic symptoms of the disease. After treatment, cleansing the blood, rigorous disinfection of the mouth with ferruginous injections, change of diet etc. there appeared classic phenomena of polyneuritis; walking was impossible; there was high-stepping, pes equino varus, great weakness of the lower limbs, with muscular atrophy and pain in the gastrocnemius on being touched. Rotular and plantar reflexes were absent; there was progressive anaesthesia of the roots of the lower hinbs and paraesthesia of the upper limbs. Tongue dirty and trembling. Hyperphonesis of the pulmonary sound and some times of the second murmur. Pulse weak, 74 times a minute. Microscopical examination of the excrementa revealed eggs of ankylostoma; they are commum amongst the insane in Brazil. The patient had a tendonous rectraction of the right leg, on account of a scorbutic ecchimosis on the popliteal region. The muscles and nerves, especially those of the inferior hinbs, did not answer to faradisation, and only irregularly to the galvanic current, mostly showing Erb's reaction of degeneration (D. R. incomplete). Extreme weakness and cachexy were noticeable: the patient weighed 22 kilos and 900 grs. He was transferred to another ward. After faradisation, galvanisation, massage, strychnine and change of diet the patient, put on weight till he weighed 43 kilos; the morbid phenomena disappeared in 6 months, leaving him almost cured.

Although the author thought of the possibility of a new affection (scorbutic polyneuritis), his observations form a valuable document in favor of modern doctrines. From his observations we may conclude that scurvy and beriberi are two different diseases caused by defficient nourishment; they were found in the same individual, mingling their symptoms, so that they seemed to be a new disease. And as their principal symptons come from the want of certain vitamines, being at

different times accentuated or aggravated, an infinity of modulations may result, so that each patient seems to have a different morbid disease.

EDUARDO MEIRELLES studied the last epidemic of beriberi in the prison, in 1915 and although he almost denies the hypothesis of its being due to the alimentation, he owns that the disease disappeared almost entirely after disinfection and after better food was given to all the patients who were treated in the Prison Infirmary.

In 1916 I was called upon to present a memento of my ideas on the etiology of beriberi in the province of Amazonas, to the General Director of the Board of Health. In this work, which by kindness of Dr. CARLOS SEIDL was published in the "Jornal do Commercio", I tried to prove by my observations that beriberi showed a tendency to disappear since the suppression of the importation of rice from the East (polished Rangoon and Carolina rice) and by the diminished importation of flour and beans, which only used to arrive after a long journey and the nstay in damp and hot storehouses.

I take this opportunity to transcribe the table of mortality due to beriberi in Manaos from 1897 to 1917.

Years	Deaths due to Beriberi	Total Montality
1897	75	
1898	100	
1899	99	
1900	137	
1901	53	
1902	59	
1903	103	
1904	204	
1905	175	
1906	121	
1907	63	1429
1908	119	1692
1909	83	1603
1910	149	2196
1911	135	2328
1912	80	1915
1913	61	1681
1914	20	1305
1915	12	1204
1916	7	1595
1917	3	1070
1918	1	2170

Formerly, there were often on the ships navegating the Amazonas river, cases of death among individuals who felt ill and embarked for the capital. With the aid of data supplied by the Board of Commerce, I included, in the above-mencioned relatory, comparative tables of the importation of the cereals, which form a great part of the food of the inhabitants of those regions, and of the cases in the capital and on board the vessels coming from the interior which were registered by the Board of Health of the Port. In this paper, I called the attention of the Government to the necessity of a legislation regulating the improvement of rice, similar to that which exists in the Phillippine Islands. Convinced, as I was, that our beriberi is the same as Eastern beriberi, I tried to prove by documents from SCHAUMANN, Japanese and American authors, that what I

Year	Importation of Rice		Beriberi Obituary in the capital	Obituary on board	Total
1911	29.998	bags	135	54	189
1912	24.179	«	80	17	97
1913	12.189	«	61	20	81
1914	5.640	«	20	2	22
1915	3.639	«	12	4	16

Year	Importation of flour from Rio Grande		Beriberi deaths in the capital	Beriberi deaths on board	Total
1911	13.137	bags	135	54	189
1912	5.563	«	80	17	97
1913	2.270	«	61	20	81
1914	5.211	«	20	2	22
1915	2.191	«	12	4	16

Year	Importation of foreign beans		Beriberi deaths Capital	Beriberi deaths on board	Total
1911	77.539	bags	135	54	189
1912	85.021	«	80	17	97
1913	64.021	«	61	20	81
1914	51.610	«	20	2	22
1915	14.687	«	12	4	16

said was true, claiming the attention of competent persons to judge in the matter.

In July 1916, appeared MIGUEL COUTO'S masterly work "Lessons on Clinical Medicine" in two lessons of which the author analyses and criticizes the current doctrines.

In the same year THEOPHILO DE ALMEIDA published his thesis on Beriberi. This contains all the modern theories in vogue, and discusses the development of beriberi in several provinces of Brazil. It deserves to be better known as the ideas defended deserve to triumph.

In his historic memoirs, presented in 1917 to the Faculty of Medicine, AFRANIO PEIXOTO discusses, in clear and elegant language all the different questions of pathogeny in the Amazonas province, and shows himself a partisan of the alimentation theory. In 1917, CLEMENTINO FRAGA published a synthetic study on "Beriberi or beriberi syndrome."

In this work, FRAGA analyses the epidemics of beriberi in Bahia, from 1866 to 1916. The beriberi epidemics in St. John's Asylum, the Pentenciary, the Palm Barracks, the Hospital of St. Elizabeth are all carefully studied. For FRAGA, beriberi is endemic in Bahia, and in the places where there are many cases, it has become from time to time epidemic; for this reason he believes in the infectious etiology of the disease. For him beriberi is an illness and not a syndrome.

In 1918 ARLINDO DE ASSIS' thesis appeared in Bahia; it was written under the direction of CLEMENTINO FRAGA, and is quite a remarkable work, both because of the doctrine and the experiments and because it is probable that after having seen those experiments Professor FRAGA should have modified his opinions.

As I said before, there are very few medical men in Brazil who have studied this disease. On the other hand, since EIJKMAN'S discovery, a great many English, Japanese and Americans authours have published much valuable work on this subject.

We must remember that in the East het idea of beriberi being caused by insufficient nourishment is not a new one. Even before there were any experiments mady, the practitioners had noticed this. In 1867 VAN LEENT said: "The principal cause of beriberi is an uniform and insufficient diet of bad quality." The organism being deprived of the indespensable elements for the formation of the blood, nutrition is impoverished by degrees. WERNICH in 1878 declares: "Kaki is a constitutional chronic disease of blood formation and of the vascular system."

"The rice, exclusively used as food, is chiefly responsible for the disease, not because it is of bad quality, (as one supposes) but because it is very satisfying so that the power of assimilating other food is reduced; and however good it may be, it is not sufficient for alimentation and blood formation."

TAKAKI obtained brilliant results in the Japanese Navy. He was not however able to cope with the opposition with which he met, nor to introduce his proposed reforms in the Army.

Everyone knows how great was the mortality amongst the Japanese army from beriberi during the Russo-Japanese war (80.000) and how small the proportion amongst the marines. Round Port Arthur, there was not a single death amongst the marines of the Naval Brigade, whilst the soldiers were decimated, though both endured the same conditions of climate and temperature while feeding differently. TAKAKI'S theory was true, but he erred in thinking that the defficiency was in nitrogen. For this reason his theories were not generally accepted by his countrymen.

BRADDEN (1907) for many years sustained the theory that the cause of the disease was not the rice as an article of diet, but that there was an alteration in it by germs or spores; these spores either existed in the husk and were mixed with the rice during the process of cleaning, or they developed after the shelling.

In 1908—1909 FRAZER and STANTON

made direct experiments on men for the first time. These belonged to an agricultural establishment in Malay. 300 javese were divided in to 2 groups. To group "A" polished rice was given as principal food; to group "B" was given rice in the husk, brown rice, as principal diet. At the end of more or less 90 to 100 days, cases of typical beriberi appeared in the group "A" whilst group "B" remained quite well. If group "A" had its diet changed, the patients got well and beriberi disappeared. Several subjects from group "B" were mixed with those of group "A"; if they ate brown rice, they remained healthy, but if they ate polished rice, they presented symptoms of the malady after some time.

Thus was proved the non contagiousness of the disease and its alimentary origin. All precautions were taken, to avoid any error in the observations. At the same time, many well known scientists made the same experiments in animals and the conclusion of the etiological and symptomatic identity of the two diseases was reached.

FLETCHER in 1909 treated 123 inmates of the Kuaia Lumpur Lunatic Asylum with polished rice and 123 with rough rice; in the first group there were 43 cases of beriberi, in the second none at all. The two groups changed from one building to the other but no case occurred in those who ate brown rice. Then they changed the rice food of each group. No new cases arose amongst the first group but there were cases amongst the second group, then feeding on polished rice.

In the Health Bulletin nº. 12 published by the Department of the Interior, of the American Government in the Phillipines, the same results were obtained amongst the American scouts. There were very many cases amongst these soldiers. In one year there was a return of 600 cases. After giving them brown rice, the disease diminished and there were no new cases. The Governor then decreed that no polished rice was to be used in the Government Institutions.

And says the report, the disease disappeared everywhere. It was then proved that the few cases that occurred ensued from some department not having exactly obeyed the rule about polished rice. In this same Bulletin the government asked all the better classes to use unpolished rice, to give an example to the uneducated classes. The Board of Health proposed a tax of 4 centavos on the kilo of polished rice, so as to exclude the poor from making use of it; if the richer people used it there was not so much danger for them as they have other foods sufftciently nutritious to prevent the rice from harming them.

In 1913 STRONG and CROWELL published the result of their investigations in Spanish language and this work is so rigorous ly scientific that I consider it as the greatest and most definite argument with regard to the alimentary etiology of beriberi. The principal value of this work lies in the record of experiments on man, investigating the problem from all points of vue an thus completing the studies of FRAZER, STANTON and FLETCHER. Convinced that MANSON, SCHEUBE, LE DANTEC, MARCHOUX WRIGHT, CASTELLANI, SHIBAYAMA did not bring forward sufficient proofs of their theory of infection, STRONG and CROWELL tried to reproduce the illness, excluding the influence of specific micro-organisms. They acknowledged the importance of the studies of *polyneuritis gallinarum* as the experiments on the birds were very clear and elucidated manv problems in the aetiology and the cure of beriberi. They thought however that without identical experiments made on man, nothing absolute could be positively affirmed from a scientific point of view. These wonderful experiments were made in the prison of Bilibid in Manilla, in which the hygienic conditions are almost ideal. With the Governor's permission, a certain number of criminals under scntence of capital punishment were chosen for the experiments.

To these men, STRONG and CROWELL explained with the utmost frankness, in the

Philippine dialect, the nature and danger of the experiments which would be made on them. They were told that the disease might end fatally for them, but that all that they were required to do was to eat certain kinds of rice. In compensation, they would receive as much tobacco and as many cigars as they liked. Twenty-nine offered themselves voluntarily. They were made to sign a document declaring that they had offered them selves of their own free will, and promised to continue until the end. They were divided into 4 groups: three of these groups were taken to one side of the building and the 4th group to the other side.

The sanitary conditions were excellent, and extreme vigilance was exercised to prevent the men receiving the slightest portion of food, outside that which was given them for the experiment. Their food was prepared by a special cook, and carefully weighed out for them at each meal. When cooked, the the food was served out to them in equal portions. Each person received his ration in a bowl separated for him, with his number on it. For each meal, fresh rice was cooked and was given hot.

Diet used.

Slight repast called little breakfast:— Bread — 100 grs. — Coffee 500 cc. Sugar 15 grs. First Breakfast: Rice 300 grs. — Bacon 50 grs. Second Breakfast: Rice 300 grs. — Onions 100 grs. — Butter 15 grs. Third Breakfast — Rice 300 grs. — Bread 100 grs. Fourth Breakfast — Rice 200 grs. — Bread 150 grs. — Bacon 30 grs.

First Dinner: Rice 50 grs. — Onions 150 grs. — Butter 20 grs. Second Dinner: Rice 300 grs. — Bacon 50 grs; Third Dinner: Rice 300 grs. — 150 grs.

This diet was given alternately to groups I, II and III. On the ninety — seventh day, and on the eighty first day for the third group 30 grs of codfish and 100 grs of potatoes were added, served with 300 grs of rice and alternated with the other food. The diet was the same for all except in the quality of the rice used.

The plan for the experiments was as follows.

Group I. — Polished white rice with meal from the rice husk.

Group II. — White rice and alcoholic extract of the cuticle.

Group III.—Polished white rice.

In spite of all, after some days some of the prisoners would not eat rice mixed with meal of the cuticle or prepared in a manner that made their food palatable. For instance Group I would not eat rice with meal and they were given dark rice (brown rice not polished) which is called *pinawa* in the Philippine Islands. The extract of bran was made by Dr. FELIX OCKSON of the laboratory of the College of Medicine by the process of VEDDER and CHAMBERLAIN.

These experiments are worthy of being copied in totum but unfortunately it would draw out our paper to an undue length. We will therefore make a synopsis and give the conclusions.

STRONG and CROWELL drew the following conclusions.

1. Beriberi in these men was due to their diet and therefore the disease has a dietic origin.

2. Beriberi appears when there is a want in the food of one or more substances necessary for the normal physiological processes of the organism.

3. When these substances are not found in the diet beriberi ensues. The substances exist in brown rice and in smaller quantities in the alcoholic extract of rice-bran, so that when one of these things are added to a diet physiologically appropriate, it prevents the appearance of beriberi symptoms.

4. In some cases though this substance forms part of the diet, the latter not being varied and being limited in kind, the patient loses his appetite and weight. These patients may present beriberi symptoms. The symptoms can however be stopped by changing the diet or adding other nutritive foods to it.

5. It is evident, say STRONG and CRO-

WELL, that the disease is not infectious but due to diet.

7. It is not probable that the patients could have been infected through the food, as it was always freshly cooked at a temperature to which only a sporogenous microbe could resist.

Besides this, if the infection had been introduced by the food, it would have been the same in all groups.

8. None of the rice used fermented, a fact which excludes the action of certain bacteria.

9. There is no proof to suggest the idea of beriberi being an infectious disease and it is certain that in the Phillipine Islands beriberi is due to a prolonged diet in which certain substances necessary to the normal physiological functions of human organism are wanting.

10. The disease studied was really beriberi as proved by the autopsy of the patient who died.

11. More observations should be made on the chemical properties and nature of the substance which prevents the disease; from a pratical point of view however the etiology is known; prevention and cure are easy.

12. To prevent and cure the disease in man, nothing is wanted but liberal nourishment of the best quality adapted to the physiological necessities of the organism.

COBB, in a report on North Borneo written in 1916, says that beriberi has been long known there, but there has never been a serious epidemic. Those who are most liable to contract beriberi, are the poor, opiumsmokers and coolies employed in the rubber and tobacco plantations. A minutious investigation of the slight epidemics among them revealed that they were always arising from deficient nourishment and in two cases from the use of Saigon rice. COBB speaks of the great trouble there always is in the East to get the natives to use unpolished rice. The disease could have been eliminated. Everything depends on the food of he working classes.

In 1917 CHICK and HUME made an interesting communication to the LONDON SOCIETY FOR TROPICAL MEDICINE AND HYGIENE about beriberi amongst the English troops during the siege of Kut-el-Amara. The disease entirely disappeared with the change of food.

The British, who at first received a ration of white flour, afterwards received only a third or a half of this ration which was substituted by barley-meal or coarse wheat flour. They were protected against scurvy, being fed on ample rations of horseflesh. The Indian soldiers were supplied with cereals and beans rich in anti-beriberi vitamines. The Indians refused the horseflesh, and as there were no vegetables nor fruit in the region, they became an easy prey to scurvy. The authors made the following deductions:

1. To prevent beriberi, it is imperious that the germen (embryo) and the bran should not be separated from the flour used for making bread and biscuits for the soldiers on active service.

2. This is still more important when the troops are far away from all resources of fresh food, and live chiefly on tinned food which is deprived of vitamines by sterilisation at high temperatures.

3. To prevent scurvy, if there be no possibility of their obtaining fresh fruit and vegetables, they ought to be given seeds of leguminous plants which are already beginning to sprout.

It has been clearly proved by many of these who have studied the matter and by many experiments made on man, in the Philippines and in Malacca, that the origin of beriberi lies in the alimentation. One more argument must be cited here. I refer to the efficacious cure with Katjangidjo. This kind of beans from which HUSCHOLFF POLL extracted an acid substance which he calls X-acid, is not only useful to prevent the disease but also to cure it.

Vitamines are still very difficult and expensive to extract but the alcoholic extract of rice-bran can be given as a curative of

great efficacy. The celebrated case described by VEDDER ought to be known, and we herewith give a summary of it:

CIRILLO TAQUINEZ entered the General Hospital of Manilla on September the 5th. On the 9th. VEDDER found him in bed suffering from an intense dyspsnoea, breathing with the greatest efforts. The pulse was 150 a minute; respiration 45 and the heart-beat violent. It was the typical history of a chronic case of beriberi with acute cardiac exacerbation. All the muscles were attacked and extremely painful when touched. For several days the patient had not eaten, on account of his vomiting. The assistant-doctor thought he could not live through the night and as the case was so serious, resolved to give it over to VEDDER. The latter ordered alcoholic extract of rice-bran which was given at 11 o'clock in the morning of the 9th. Fortunately the patient retained it. In the afternoon he got better, his pulse fell to 80 and respiration to 30. The next day he had a fresh exacerbation and was again given the alcoholic extrait of a kilo of rice-bran. The dose was administered daily for a fortnight. The cardiac symptoms did not reappear; the hyperaesthesia of the muscles diminished a great deal, he did not vomit any more and regained his appetite improving greatly in appearance though the paralysis remained.

Similar results were obtained in other cases so that it was clearly proved that the disease was human beriberi (dropsycal form), easily cured by this process. In the dropsycal form and for cardiac trouble this remedy is therefore useful.

For the paralysis dependant on the lesion of the nerves and muscular fibres there exists no sure remedy; it can only be cured slowly, and by intelligent and careful dieting of the patient. This is easy to be understood as the restoration of important elements in the structure and the restitution *ad integrum* of a cell, such as the striated fibre or neuron can only be effected with comparative slowness and only if the destruction be not definite and total.

An objection of great scientific value against our arguments was emitted by the well known Brazilian doctor MIGUEL COUTO, in his book published in July 1916. He cannot believe that rice is the principal factor in causing beriberi, as was proved by BRADDON at he Congress of London, since rice does not cause beriberi as often in Brazil as in Asia and Oceania; he resumes all his objections in the following conclusions reproduced here textually:

As to symptomatology and as to clinical forms, evolution, anatomic substratum our beriberi is the same type as asiatic beriberi but it differs in aetiology, as is proved by the following facts.

1. The rice consumed by the people in the North is *the same* (1), both raw and cooked, as that eaten in the South; we have no reason to believe that it differs much in the quantity consumed, yet in the North beriberi is endemic and abundant and in the south almost unknown. (2)

2. In Brazil beriberi is more prevalent, in some States, and in these it is found in certain localities and in certain buildings, where there are agglomerations of human beings, while on the other hand the rice eaten by all is the same.

3. Rice is one of the staple articles of food in our country; it is not used so as to exclude other foods but it is eaten by all classes, and the vitamine esse quid is wanting to all, whilst some people are attacked with beriberi and others not.

4. Whilst there must be a receptive organism for infections to develop, intoxications have a universal action; but of all the people in our country who eat rice, some contract beriberi and others not.

Note 1. The italies are mine. Certainly Professor COUTO did not know that most of the rice eaten in the north comes from Rangoon and the Caroline Islauds.

Note 2. In a previous paper, I noted that the india-rubber workers eat only, for months at a time, dried meat, flour, rice and beans all of which are imported, and make long journeys also remaining stored for some time.

5. Traditional observation has shown that the best remedy for beriberi is the removal of the patient from the centre where he contracted the disease; a half-dead patient embarks here and arrives quite well in Europe or in Buenos-Ayres; this feat is not compatible with the idea that he has had an alimentary intoxication, because, even changing his diet, he would have died if he had been left where he contracted the disease. (1)

6. Traditional experience has also proved that, even if the patient be cured, he may have a relapse, if he returns to the place where he fell ill, although he may eat no rice; whereas, if he remains away, there will be no relapse.

7. The rice furnished to the Navy is the same for one and all, and some vessels are more devastated than others, though the same store purveys rice to all. It is certainly the prisoners in the gaols who suffer most.

8. The rice sent to the troops in the Paraguayan war was the same as that consumed by the civilians who admired the valour of their compatriots from afar; these latter enjoyed good health while the soldiers were victimised in numbers by this fell disease. The same will be said by the Japanese, who lost 80, or (according to some opinions) 150 thousand soldiers in the with Russia war, who also ate the same rice as those who stayed at home.

9. Finally, my patient always eat about the same amount of the same kind of rice, and only fell ill once, whilst all the rest of his family ware in the same condition.

Carried away by the logic of these arguments MIGUEL COUTO concludes thus:

"The disease by which our patient is attacked, is the same as the disease reigning in India and Japan under the name of beriberi. PATTERSON and SILVA thought they had discovered it in Bahia in 1894; the cause of the former is now discovered but that of ours is not. We erred therefore in the interpretation. Our beriberi is not beriberi."

We admit the plausibility of these arguments, until it was found that it is not only polished rice that causes the illness. Now we know that all cereals contain vitamines which can be destroyed by various conditions, as is proved by the experiments of SCHAUMANN, of SHIGA and others. The food may seem to be perfectly good and yet for multiple causes the protective substance may have been destroyed, and thus the food may have lost its most important biological qualities.

Latterly, new studies have shown that prolonged sterilisation deprives it of the properties indispensable for perfect nutrition and to the development of the organism.

SCHAUMANN saw beans enclosed in glass jars in Hamburg in a laboratory, looking as if they were quite perfectly preserved, but they had lost the power of curing *polyneuritis gallinarum*, which the same kind of beans possess when fresh.

ARLINDO DE ASSIS proved that beans sold on the Bahia market were defficient and deprived of their preventive substance against experimental beriberi.

It is a fact that all the beans imported in the State of Amazonas for the use of the men employed on rubber plantations come "roasted" as a preventive against weewils.

The observations made by JAYME SILVADO in 1907 do away with the objections made against the superior quality of the food furnished to the Navy.

A more or less uniform diet kept on for a long time, becomes defficient; the first sign the disease is want of appetite; the defficiency grows from day to day, provoking all the most grave symptoms. As a proof of this, we need only cite the beriberi attacking the British troops at Kut-el-Amara. For this reason, in the beginning the patients may be cured by sending them on a journey, thus changing their food entirely.

In the East the problem is very difficult on account of the numerous poor people, but here no such dificulty exists. Except

Note 1. Observations mado by AUSTREGESILO, MEIRELLES and myself (in Amazonas) prove that is is possible to cure beriberi *in loco*, if the symptons are not yet fatal.

in the indiarubber plantations, in Brazil, beriberi is a disease of barracks, ships, asylums, penitentiaries and hospitals. The purveyor is the first cause. He ought to be, permit the expression, eliminated by decree.

In the rubber plantations, the disease became rarer lately, owing to the war and the consequent depreciation of Brazilian indiarubber, as was proved to me by indisputable statistics. Through the agency of an intelligent and infatigable propaganda made by the Sanitary authorities in the North, it might be eradicated.

VEDDER, STRONG and CROWELL ask for the education of the people in the East and the Health Bulletins of the Philippine Islands began a propaganda for this end by exposing the matter in simple language. VEDDER speaks of the want of energy in the Eastern nations and in their persistance in their ancient customs. And what people are more like the Orientals, with their fatalism, their indifference and resistance to modern ideas than our people of the North?

Measures ought to be taken. Already in 1916 I asked the Government to decree laws to prevent the exclusive use of polished rice, or at least to a put a prohibitive tax on it, so that only rich people could use it.

Mandioc flour ought to be rigorously fiscalized, so that it be only used in theybest state of preservation. But then my ideas on alimentation myust be confirmed. The disease and its causes must be discussed and in these debates the voices of the princes of science must be heard at the side of the murmur of their humble disciples. And if the alimentary theory be true, the diffusion of these ideas will ensure the carrymg out of the least costly and most necessary propy lactic needs of the country.

Explanations of the Plates 34—36.

Plate 34.

Fig. 1. Pigeon, nº. 8. — Food: polished rice sterilized at 120 C. — Paralysis of the right wing. High stepping.

Fig. 2. Pigeon nº. 9. — Food: At first raw polished rice, afterwards rice sterilized at 120. — High stepping. Paralysis of the legs and wings.

Fig. 3. Cock nº. 10. — Food: Raw polished rice. — Weak legs. High stepping.

Plate 35.

Fig. 4. Cock, nº. 10. — Food: raw polished rice. Difficulty in walking, paralysis of the extensors.

Fig. 5. Hen nº. 15. — Diet: national raw polished rice. — Paralysis of the legs on the 30th. day of observation.

Fig. 6. Cock nº. 16. — Diet: polished sterilised rice. — Paralysis on the 17th. day. — Died on the 19th. day.

Plate 36.

Fig. 7. Cock nº. — Diet: Indian corn autoclaved during an hour at 120. — Beginning of paralytic phenomena.

Fig. 8. Cock uº. 13. — Diet: Indian corn sterilised in the autoclave for one hour at 120. — Paralysis of the extensors almost preventing it from walking.

Fig. 9. Diet: Sterilised Indian corn. Food administered by gavage to prevent death from ination. — This state lasted for thre days before death.

Schistosomum Mansoni and Schistosomatosis observed in Brazil

by

ADOLPHO LUTZ, M. D.

(With Plates 37—43).

Biology and evolution of the trematodes belonging to the genus *Schistosomum* were but little known until a few years ago, but since 1911 several papers were published which gave the solution of the most important problems for four species. I report here the results of my investigations, directed mainly to repeating and controlling the recent experiments in connection with the only species known in Brazil, yet also to determining the local species of the intermediary host and the conditions of infection in this country. In order to give more pratical value to my paper and to help the orientation of the readers, I decided to recall briefly the most important facts in connection with the parasitism of all the schistosoms and particularly the species *Mansoni*. Of litterary references I only give the most important and those in closer connection with my paper; for the rest I may refer the readers to the list given by LEIPER (1915) which is easily obtained and very extensive, containing 529 numbers.

I shall first consider the historical and and geographical part of our knowledge concerning the genus *Schistosomum* and the diseases caused by the same.

Schistosomatosis or Bilharziosis in Africa.

The troubles caused by the African species of *Schistosomum* were known long before their cause and existed in remote periods, as the examination of mummies has proved. Since the invasion of Napoleon until our time they were very common there. In this classical field of observation the parasites were discovered in 1851 by BILHARZ who called them *Distoma haematobium*, on account of their inhabiting the blood vessels. He was well fit for such studies and recognized that this very peculiar trematode was specially caracterized by separate and bimorphic sexes, a quite new and unexpected fact. The name *Schistosoma* WEINLAND, now generally latizined *(Schistosomum)*, seems to apply to the male whose body has a fissure, opening a longitudinal furrow in which the female is carried. Hence the name *Gynaecophorus*, given by DIESING in 1859. In the same year COBBOLD introduced, in honour of the discoverer, the name *Bilharzia* which, as well as the derived expressions *Bilharziasis* and *Bilharziosis*, was commonly employed.

(BLANCHARD stated that WEINLAND'S name was published on August 5th 1858; the one of DIESING, proposed in a session of the Academy of Science in Vienna, was published Oct. 21t and COBBOLD'S name Jan. 20th, 1859; therefore he concluded that WEINLAND'S name ought to be accepted. (LAVERAN & BLANCHARD. Les Hématozoaires II, p. 40). This name, formely little known, is generally used in the later litterature).

Already the first observations in Egypt showed that the lesions might be localized in the urinary system or in the intestine or in both, while the eggs found in them had either a terminal or a lateral spine. As both processes were very common, they were often combined in one patient who then furnished both species of eggs. Therefore the general opinion went on attributing both processes to the same parasite (although the idea of a double origin was very soon suggested). This was due mainly to some observations, always brought forward in the discussions and apparently convincing. Today, however, it is evident that, either in the observation or in the interpretation, there occurred some error, not at all unlikely to happen. This unfortunate accident proved an enormous drawback for the right understanding, of these infections.

The first observation of a focus where only eggs with terminal spines ocurred, was made by HARLEY in the South of Africa. He thought the parasite was altogether different and gave it the name: *Distoma capense* (which, in my opinion, ought to be still used for that form). In 1912 BOUR found in Mauritius 34 boys voiding Sch. eggs with their urine. He did not mention the position of the spine, nor eggs in the stools. Many observers found both forms of eggs in Tunis. In the Soudan. in the lake territory and generally in hot regions with plenty of, more or less, stagnant water, the disease of the urinary system is common. It is also found in the islands of Madagascar, Nossi-Bé and Réunion. The intestinal form is often found together with the other and aparently alone in the Belgian Congo.

Schistosomatosis in other continents.

Schistosomatosis localised in the urinary system has been found in many endemic foci of Asia. As for America, it is quite certain that there never has been an endemic focus of this form; a few cases reported were apparently only seen in people, who had arrived or returned with an infection contracted in other continents.

It is remarkable that in tropical America which imported so many slaves, the disease of the bladder never took root, while it becomes every day more evident that the intestinal form is widely spread in the West Indian Islands and on the South American continent. It is certain that both forms have been imported over and over again but only one spread. This is now easily understood, as we know that the possibility oi localisation depends of the kinds of fresh-water shells found in the place.

Schistosomum haematobium BILHARZ includes two species.

The idea that two different species were united under the name *Sch. haematobium* was again sustained in 1893 on the base of observations made in America. The fact that in 1876 SONSINO had found a second species (*Sch. crassum*) in cattle and still more the discovery of the *Sch. japonicnm* in 1903 helped to sustain this supposition. Iu 1907 SAMBON introduced the name *Sch. Mansoni* for the American species; it was generally accepted, though this form ought to have kept the name *haematobium*, the other having been distinguished nnder the name *capense* HARLEY.

The double value of the species was denied by several authors and principally by LOOSS who tried to explain the differences of the eggs by quite unconvincing arguments. It is strange that this author, who so violently sustained the unity, should at other times have separated new species and even gene-

ra with no better arguments, However, nowadays this discussion has lost all importance, as the double nature of the ancient *Sch. haematobium* is generally recognized.

There are now six more species known, all of which have different eggs, of one form only).

Some authors claim to have found eggs of both forms in the intestine and in the urinary organs. Excluding evident mistakes, these observations are altogether rare and mostly old; they refer to double infections of long standing and several explanations have been given for the abnormal localisation in these cases. In America a great number of observers failed to find a single egg with terminal spine amongst thousands with lateral ones; it must however be remembered that a lateral spine in certain positions may appear terminal, unless the egg be rolled. As seen in figs. 10 and 11, they may have an obtuse prolongation situated at the more distant pole and very different from a spine. The localisation in the urinary svsteme causes different and very striking symptoms, while those of the intestinal form may easily pass unnoted. Already WUCHERER searched for Schistosomum eggs in cases of haematuria, observed in Bahia, and failed to find them, though in the same place the other form occurs frequently. In a great number of urine examinations which I made during the space of 38 years, I failed to find a single egg of *Schistosomum* though their form was well-known to me, a fact which, combined with the experience of many of my colleagues, is sufficient to exclude the existence of this fluke in the more known parts of Brazil. On the other hand I examined the stools of about twenty patients of *schistosomatosis* of the intestine, acquired in the north, in some cases very often, without finding one egg with terminal spike. My observations are confirmed by those of Dr. OSWINO ALVARES PENNA, who also observed some of these cases and many others, and agree with the results obtained in the West Indian Islands, in Dutch Guiana and in Venezuela.

In another place, I shall explain the characteristics which distinguish the two species. This paper is limited to *Sch. Mansoni* SAMBON, which is the only one found in Brazil, and, indeed, in the whole of America, with the exception of imported cases. In the latter sometimes *Schistosomum* has been found located in the urinary system; this generally happens in those of African origin. With the increasing emigration of Asiatics, there may appear cases of Asiatic origin, due to *Sch. haematobium* or even *japonicum.* The transmitter is different from all our indigenous mollusks, so that the importation of the Japonese parasite need not be feared.

O Schistosomum Mansoni na America.

Since the year 1902, cases of *Sch. Mansoni* have been observed in America or in persons there infected. The first centres observed or pointed out by the patients were in the West Indian Islands. Here we may quote the cases of MANSON (1903, infection in Antigua or in one of the other West Indian Islands), of LETULLE (1903 in case of Martinique), MACDONNELL (1905, case of Culebra), LAHILLE (1906, ibid.). In Porto Rico which is an important centre, GONZALEZ-MARTINEZ observed and published the first cases in 1904. Later on, ASHFORD, KING and GUTIERREZ IGARAVIDEZ often found eggs, while they were studying endemic ankylostomiasis. The studies of these authors, as well as later work, prove that Martinique, Guadeloupe, Vicques, Culebra, Portorico and Cuba must be counted amongst the principal centres (v. HOLCOMB, 1907).

In medical literature, we find unexplained cases of urinary schistosomatosis with the eggs belonging to it, observed by CORTEZ in 1905 in a native of Guatan, Guatemala. Dr. BUTLER is also quoted as having observed a patient from Porto Rico in San Juan, who evacuated two eggs with terminal spine in his urine, without knowing where he contracted the disease. As a great number of patients have been examined in Porto Rico by competent physicians there is little

probability of that place being an endemic centre of this form of Schistosomatosis; the doctors of this island are unanimous in recognising the frequence of *Sch. Mansoni*, and the lack of eggs with terminal spine.

Other cases, observed in the zone of the Panama Canal by ZEILER (1900), appear in the literature without information as to the zone of infection. Amongst these one case is mentioned where the two forms of eggs coexisted with the two forms of infection. If the case be authentic, I an inclined to consider it as an infection, imported from another continent.

In the South-American continent, cases were reported from Dutch Guiana (FLU 1908), Venezuela (BREM, JESUS RISQUEZ), Columbia (BATES) and Peru. According to a recent publication by JESUS RISQUEZ, it seems that the infection is very common in Venezuela, as he found it in 25 % of the autopsies made in Caracas, which means an intense focus. He also studied the pathological anatomy.

In 1905 BLUMGART of New York observed *Sch. Mansoni* in a German, probably infected in Brazil, as he had lived in that country for 7 years.

In 1908 and 1909 Dr. M. PIRAJÁ DA SILVA made a series of communications by which he proved the frequency of intestinal Schistosomatosis in Bahia. He tried to characterize the parasite as a different species which he called *Sch. americanum*. He also described a cercaria found in Bahia under the name of *Cercaria Blanchardi*. Subsequent studies showed that as well its general form, as its host. a large *Planorbis*, agree with the supposition that he was dealing with the cercaria of *Sch. M.* JOHN MILLER and several other writers also recorded observations from Bahia.

As for Rio de Janeiro Sch. eggs-with lateral spines were found in stools by FARIA, in tumours of the rectum by VIANNA and in the pancreas by D'UTRA. Though these and other observations were made here, the anamnesis, when known, pointed to an infection in the states of the North of Brazil. The same may be said of many cases observed by PENNA and others where the eggs were discovered while looking for Necator eggs. The cases ocurred mostly in sailors and marines who were infected before entering the schools, mostly be bathing in ponds containing fresh water shells. Some of the infections had taken place many years ago. The patients hailed from the states Bahia, Sergipe, Alagoas, Pernambuco, Parahyba and Rio Grande do Norte which are undoubtedly infected zones. To these we may add Goyaz where NEIVA, on one ocasion, observed characteristic eggs. Infection probably also existed in a place in Ceará, in a swamp near Obidos and in a "lagoa" in Espirito Santo. The infection is so little characteristic, as to be recognised only when either a microscopical examination of the stools or a careful post mortem examination is performed.

It is only due to the stool examinations, now commonly made for the research of *Necator* and *Uncinaria*, that the infection was discovered in so many new centres. The indispensable conditions for the existence of endemic centres are, on one side, a hot climate, on the other, the presence of a species of Planorbis which is apt to become an intermediate host. In Brazil *Pl. olivaceus* and at least two more species are easily infected, but they do not seem to exist in Rio de Janeiro or to the south of the city.

In 1916 the institute sent one of its servants, named THEOPHILO MARTINS, to Aracajú. He verified the infection in several persons who used to bathe in ponds where *Planorbis olivaceus* was common. Of these he brought a great lot which I used for infection. We also received specimens from our colleagues Drs. PIRAJÁ and OCTAVIO TORRES and used them for experimental infection. In the last lot I found some naturally infected specimens which furnished living cercariae,

Recent observations made in the North of Brazil.

A special commission was sent by this institute in the second semester of 1917, in

order to investigate the local conditions in the states Rio Grande do Norte, Parahyba, Pernambuco, Alagoas, Sergipe and Bahia, known as centres of intestinal schistosomatosis. The commission, composed of Drs. ADOLPHO LUTZ and OSWINO PENNA and two laboratory servants, found cases in all these states, especially in Pernambuco and Sergipe where they remained longer. They verified that *Planorbis olivaceus* is limited to the states Bahia and Sergipe; more to the north the intermediary host is evidently a smaller species, described as *Pl. centimetralis* by LUTZ in his monograph of the South American Planorbis, published in the Memorias do Instituto Oswaldo Cruz, V. X, F. I, 1918. Quite to the north of the zone, crossed by the commission, appeared another species, *Planorbis guadaloupensis* SOWERBY which is the intermediate host of Sch. M. in Venezuela and probably also in all the Antilles where it exists. Of the last two species no infected specimens were found which, in the case of *centimetralis*, was probably due to the unfavourable season. I obtained the experimental infection of spemens belonging to *Pl. guadaloupensis*, received by the kindness of Mr. FABRICIO CALDAS DE OLIVEIRA in Maranhão. The infection of *centimetralis* has not yet been completed, though penetration of the mtracidia was observed on several occasions. Spontaneous infection of *Pl. olivaceus* was observed once in Sergipe and three times in the state Bahia.

Later informations induce me to include Minas Geraes among the states infected with Sch. M. I learnt from Dr. HENRIQUE VILLELA that Dr. ABEL TAVARES observed severals patients in Bello Horizonte, voiding eggs of Sch. M. and probably infected in that region. Dr. PENNA also saw a case coming from the zone of the river Pomba in Minas.

This agrees with anterior observations of LUTZ showing the occurrence of *Pl. centimetralis* in Retiro near Juiz de Fora (Minas). Recent observations made by CHAGAS indicate that *Sch. M.* also occurs in the Acre territory; thus actually all the states north of Rio may be considered as infected.

I conclude this chapter with a recapitulation of the most important dates:

1851 Discovery of *Distoma haematobium* in Egypt by BILHARZ.

1858 WEINLAND proposes the genus *Schistosomum.*

1864 HARLEY calls *Distoma capense* the South African blood flukes having eggs with terminal spines.

1874 SONSINO discovers the *Sch. crassum* in cattle but fails to find the intermediary host of the human species.

1876 COBBOLD fails to find the intermediary host.

1888 ALLAN indicates infection through bathing (also BROOK in 1897).

1892-1903 Imported cases of *Sch. haematobium* observed in the United States and in Panama.

1902-1903 MANSON, LETULLE, GONÇALEZ-MARTINEZ and others begin to observe eggs with terminal spine in the stools of patients infected in the Antilles.

1905 BLUMGART in New-York observes eggs with lateral spine in the stools of a person having spent 7 years in Brazil.

1907 SAMBON introduces the name: *Schistosomum Mansoni.*

1908 First communication of M. PIRAJÁ on the occurrence of *Sch. M.* in Bahia.

1911 First communication of FLU on *Sch. M.* in Dutch Guiana.

1911-1913 Discovery of *Sch. japon.* and its biology.

1915 The LEIPER commission studies the evolution of human blood flukes.

1916 LUTZ studies the evolution of *Sch. M.* in the brazilian *Planorbis olivaceus.*

1916 ITURBE and GONÇALEZ report that the host of *Sch. M.* in Venezuela is *Planorbis guadelopensis.*

1917 It is shown that some of the brazilian foci depend on *Pl. centimetralis* and that *Pl. guadeloupensis* also occurs.

1918 The host of *Sch. Spindale* is discovered by GLEN-LISTON and SOPARKAR.

Descriptive Part.

The genus Schistosomum.

(Figs. 1–8),

The genus *Schistosomum* comprises different species of blood flukes which posses an oral and an abdominal sucker and were therefore at first included in the genus *Distoma*, now usually latinized *Distomum.* This genus is now subdivided in many others forming the family *Distomidae. Sch.* is distinguished from most of them by having the two sexes distributed on two individuals instead of having them united in one. Another characteristic is the *habitat* in the blood vessels to which these flukes are well adapted.

Similar characters are found in a few more genera, allied and only recently separated. They form a sub-family which may be called *Schistosominae.* I quote the genera *Bilharziella* and *Ornithobilharzia,* observed in birds 4).

Sexual dimorphism is very marked in *Schistosomum.* The male has a flattened and rather wide body, well developped muscles and pointed skin scales which assist it in holding its place ayainst the blood stream; the female has a filiform body like a nematode; her muscles are weak and the scales are either absent or very small,

After the cephalic part the body of the male widens out, though the lateral parts are not flat but inwards curled, thus constituing the *canalis gynaecophorus* which generally contains one, rarely two or more females. The genital system is reduced to a subdivided *testis* (sometimes followed by a *vesicula spermatica)* and an excretory duct; there is no *cirrhus.*

The female has a *corpus vitelligenum,* an ovary and a long tube corresponding to *uterus* and *vagina.* A small segment which contains the first (and sometimes only) completely formed egg, may be called *ootype.*

In both sexes no pharyngeal or oesophageal bulb exists and the intestine is first double and afterwards simple, in varying extent.

(There are three species living in man and five more found in domestic animals, all of them easily distinguished by their eggs. The human parasites also occur in apes and rodents (spontaneous or experimental infection), while *Sch. japonicum* is also found in most of the domestic animals).

Description and differential caracters of Schistosomum Mansoni.

(Fig. 1–8, 15.)

The anatomy and morphology of *Sch. M.* were studied by the discoverer and also by LEUCKART, R. BLANCHARD, CHATIN and FRITSCH before 1888; the results were discussed by R. BLANCHARD in 1889 and by LEUCKART in the second part of his classical treatise, in collaboration with LOOSS. The problem might have been practically exhausted, if the authors had worked on one species only, (and not with a mixture of two) or only on the form found in the urinary system. For this reason the descriptions have only a relative value and ought be corrected for each of thetwo species, confounded under the name *Sch. haematobium.*

The differences were already indicated by PIRAJÁ, FLU, HOLBORN and other authors who studied the american species, but, as they had no occasion to compare fresh specimens of the other species, the value of some differential caracters might have remained somewhat doubtful; but LEIPER who ultimately had occasion to compare both species in Egypt, quite confirmed the correctness of the most important characters

which make the distinction safe and easy. I also found in our specimens all the caracters which he attributes to the intestinal form of Egypt; this is undoubtedly identical with *Sch. Mansoni.* As shown by a recent communication, GONZALEZ MARTINEZ also came to this conclusion.

The genus characters common to both species are the following:

Localisation in the blood vessels and alimentation by blood. Existence of two sexes; males with *canalis gynaecophorus*, nematoid females. Eggs without lid, containing a miracidium when voided. Penetration of the miracidium in a freshwater mollusk followed by formation of sporocysts and cercariae which emigrate spontaneously. Cercariae without pharyngeal bulb, with bifid tail and large glandular cells, infecting the ultimate host by boring through the skin.

For distinguishing the two species the following apparently well established differences are sufficient:

Haematobium.

♂ *Testis* showing 4–5 lobes.
Anterior *Vesicula seminalis.*
♀ *Ovary* in the anterior half of the body.
Ripe egg with terminal spine.
Uterus long, with many eggs.
Corpus vitelligenum short in the caudal forth.
Coecum short.
First stages in *Physopsis.*

Mansoni.

Testis with about 8 lobules.
No *vesicula seminalis* to be seen.
Ovary in the posterior half of the body.
Ripe egg with lateral spine.
Uterus short, with one egg as a rule.
Dto. long. extending over the last two thirds of body.
Coecum long.
First stages in species of *Planorbis.*

Beside these, other differences have been indicated which refer to the dimensions of the body and the suckers, the distance between the ends of the coecum and the body and the armature of the skin in the two sexes, but I consider them less certain and evident. The form of the testicle and the intestine may vary a little, but not to a degree affecting the distinction of the species.

For the clinical diagnosis the form of the egg, found in the stool, and the localisation of the symptoms are sufficient.

In dead specimens the dimensions depend not only on the method of fixation, but also on anterior conditions which determine the contraction or relaxation of the muscles. The latter influe so much on the live specimens, that any given dimensions have only a relative value. Ancient authors did not distinguish and the newer observers failed to compare the two species under identical conditions. Of modern authors BRUMPT gives the following dimensions for *Sch. haematobium:* Male, length 10-15 mm., width not more than 1, female 15-20: 0,1-0,2 mm.; Pirajá for *Sch. M.*: male 12: 0,558 in the middle, female 14,5-15: 0,168 mm.. My impression is that *Mansoni* is altogether somewhat smaller than *haematobium.*

The colour varies according to circumstances and is not likely to be constantly different in the two species.

The Eggs of Schistosomum Mansoni. Characteristics of the eggs found in the stools.

The evolution of *Sch. Mansoni,* outside of the human body, begins with the eggs voided in the stools. These deserve special study as they furnish the principal means of diagnosis. It is impossible to arrive at a sure diagnosis by any other means and indeed the frequent occurrence of the parasite in South America was discovered only by constantly searching for the eggs of intestinal parasites.

The *Schistosomum*–eggs are rather large but the shell is transparent and delicate. They are, therefore, not well seen in opaque preparations and may be easily squashed. Due to the scant production they are rare

and easily escape observation, when the females are not numerous. Slight or recent infections cannot be safely excluded, as the eggs take some time to reach the intestine, when they are not altogether retained, which seems to happen frequently.

Examination is rendered easier by washing the feces, followed by sedimentation or centrifugation. With these may be combined the use of wire netting or muslin which retain the larger bodies. Thus we obtain a sediment that contains almost exclusively globules of starch and eggs of parasites which easily found.

The appearance of the egg is well perceived in our illustrations. The normal eggs (Fig. 15). are furnished with a lateral spine only and contain a perfectly visible embryo. In pathological eggs the size may be diminished while no embryo is found. One of the poles may show a protrusion, more or less considerable (Figs. 10 and 11), but this could only be taken for a terminal spine when the examination is carelessly made. As yet there has been no mention of two lateral spines. Figure 12 shows the only case of this rare anomaly which I found while examining thousands of eggs. The eggs, with or without embryo, may be calcified or encrusted with crystals. (Figs. 9,13 & 14). Recent descriptions of eggs with lateral spines have been published by HOLCOMBE and GONÇALEZ-MARTINEZ. Both have illustrations which are rather schematic, especially those of the latter. I also show a drawing from nature. The outside shell of the egg or chorion has a regular oval form, interrupted only by the apex of the spine, which is sharp and compact, whilst the base is conical and hollow.

The dimensions of the eggs are rather variable in the normal state. HOLCOMB. gives 112 to 162 for the larger axis and 60-70 for the lesser. Taken from thirty measures, GONÇALEZ-MARTINEZ indicates a medium of 147f or 67, 69. These dimensions agree better then those of the spike, of which the length, according to the first author, is 15-17, against 22,7 of the latter, and the width 7,5, but this may be due to the way of measuring. The point of insertion, according to HOLCOMBE, is distant from the nearest pole for the fourth part of the longitudinal axis.

The interior membrane (amnion or vitellary membrane) is near the other, but does not enter the spine and leaves an enlargement of varying thickness at the poles; as a rule, it is generally larger at the pole farther from the spine, which many be considered the anterior, as it is generally occupied by the cephalic extremity of the embryo. Thus the spine points backwards.

Both membranes are fine and transparent, but while the chorion is more yellowish, the amnion is hyaline; this pigment however, as in other eggs of entozoa, is derived from the fecal medium, and is absent in the eggs protected by a thick covering of mucus.

The embryo or miracidium inside the ripe egg.

The normal eggs are voided with the embryo alive and completely formed. The transparent membranes do not prevent examination, but weak powers do not allow of the distinguishing of all structural details. Using immersion lenses, the cover glass must be fixed on the slide, which may be done with paraffine or turpentine. Thus we may obtain clear images, but the outlines of the internal organs are very delicate and their superposition prevents their being seen all in one egg. The embryo is covered with cilia which in the anterior part are longer and point forwards. The bucal papilla is bare and on the rest of the body the cilia are turned backwards.

There are four tubular openings, of the excretory system showtng vibrating membranes, perceived in some extension.

The existence of contractile fibres, running in two or three different directions, is indicated by the lively movements the embryo makes before bursting its shell. They are not, however, so easily seen and demonstrated, as

might be concluded from the perusal of the litterature.

There is also a bagshaped gastro-intestinal organ, easily perceived when full of yolk granules. It communicates with the mouth by a wide tube which narrows towards the anterior end. The wall of this tube and of the sac are apparently flaccid.

On both sides of the stomach, a large glandular cell is easily seen, but I am not able to recognise in living specimens the nucleus, generally indicated in drawings. The excretory tube begins wide and narrows until it reaches the cephalic papilla where it bends outwards. When full, the glands and excretory ducts are very clear, the former may be turned inwards and cover the gastric sac. In fig. 15 glands and sac are not very full and show distinctly at the same level; the gland-cells are not granulated, as one might expect from some drawings of the embryo of *Sch. haematobium.*

I am unable to perceive clearly the nervous system, as it appears in some drawings. However, I see, under the organs just described and nearly in the centre, one large vesicular cell and a few smaller ones.

Their function seems like the one of a small balloon inside of a large one, giving to the body the rigidity and elasticity necessary for the movements. The rest of the body is formed by larger and smaller spherical masses; they are not very well defined and represent undoubtedly the germinal cells.

The outer and inner cilia only begin to move when the egg is in a liquid medium and the water penetrates by osmosis, as happens when the eggs are isolated by washing with fresh water.

Ecdysis and free life of the embryo or miracidium.

(Fig. 15—17.)

The favourable conditions for the ecdysis consist in pure water, light and a temperature near to 30° C. In Rio de Janeiro there are many winter days which are not suited to its observation and in cooler climates the natural conditions with rare exceptions are unfavourable.

The best process for observing the free life of the *miracidium* consists in washing, straining and sedimenting stools comparatively rich in eggs. The sediment with little water is poured in small glass dishes and exposed to the sun. The same result may be obtained by approaching an electric bulb, sufficiently to raise the water temperature to 30°. Even in the best of conditions, when the first miracidia appear soon, the ecdysis is never seen in all of them, probably because a certain number has not yet reached complete maturity. Keeping the rest in a cooler and less lighted place, the experiment, repeated once a day, still gives positive results in the next three or four days. Even after eight days, eggs with living embryo may be found but no ecdysis takes place.

Before the ecdysis we note the penetration of the water followed by a dilatation of the shell. The vitelline granules show lively molecular movement and all the cilia and vibrating membranes begin to play. Then we note from time to time an energetic contraction of the embryo which may turn completely inside the shell. The ecdysis is momentary and therefore easily missed. In one of my observations, it was proceded by very rapid and energetic rotation of the embryo which took a nearly globular shape.

The empty shell shows a spindle-shaped tear (which generally passes through its equator) and its aspect is quite as striking and characteristic, as that of the entire egg.

After leaving the egg, the *miracidium* stretches, taking a cylindrical form, and swims in large curves towards the light, until it is held back by the margin of the dish. With strong, oblique illumination it is easily recognized by its characteristic movement. (Confusion with certain infusoria and rotatoria must be avoided.) At a less favorable temperature the embryo becomes more pyriform and sinks to the bottom. The same happens, if several hours pass without its finding a suitable host.

Enclosed in capillary glass tubes and exposed in a refrigerator to very low tem-

peratures, the miracidia become first pyriform, then immovable and finally die after a short lapse of time, unless they are soon removed. In the latter case they may reassume their activity. They are always more sensitive to cold than the *cercariae* and this explains the fact that an infection, already started in the antennae of a snail, may be interrupted in consequence of cold weather.

Penetration of the Miracidia in Mollusks.

In order to watch the penetration I put a few snails in a deep PETRI's dish, adding water containing myracidia. A binocular microscope is very useful for this purpose but any lens with a weak power may be used. At first the miracidia continue their evolutions, sometimes passing close to the snails without taking any notice of them. Then they come nearer and nearer to the species they prefer. Suddenly, one or the other sticks on an exposed part. Others follow, but many remain refractory for a long time.

The species experimented upon belong to *Planorbis*, *Physa*, *Limnaeus*, *Ancylus* and *Ampullaria*. With rare exceptions (perhaps due to a previous contact with other species) the three last were completely left alone. The *Physa* and the redblooded *Planorbis* showed equal attraction, the adults being prefered; contrary to my expections, the bigger they were the more attraction they seemed to offer. The point of predilection is the antenna which is very well suited for further observations. The rest of the head and the postantennial region are also frequently chosen. The well exposed pallium is little attacked in *Planorbis*, somewhat more in *Physa*; in both the foot is neglected.

As shown in the drawing the miracidium on penetrating has an elongate, almost cylindrical form. Its movements are similar to those of a rock-drill or the piston of a syringe. The time required for complete penetration is 10-15 minutes; afterwards the miracidium is seen extended in the subcutaneous layer, showing its cilia but with the glands empty. Sometimes after several attempts the miracidium drops again or leaves the point of attack without penetrating.

Up to date, only four species of *Planorbis* are known as intermediate hosts. They are *Pl. Boissyi* in Egypt, *guadaloupensis* in the Antilles, Venezuela and the North of Brazil, *olivaceus*, found in Bahia and Sergipe and used in most of my experiences, and finally *centimetralis mihi*, found in the northern states and also in Minas. The other species described in my monograph of the brazilian *Planorbis* are not suitable. In some, as also in *Physa*, the penetration is normally effected, but evolution is arrested after the first stages.

The miracidia, which try to penetrate in other regions than the antennae or near to the eye, fail to pierce the skin or die shortly afterwards; at least I never found the next stages in other points, though possibly a miracidium might pass through the respiratory opening and reach the visceral part.

My studies were all made on the antennae which are easily observed and may be removed without danger to the snail. Even the detached antennae of *Physa* and *Planorbis* (which due to their epithelial cilia move like living organisms) are easily infected.

Successful and unsuccesful attempts to penetrate produce, in the antenna, a local reaction caracterised by swelling and hyperaemia, which is easily appreciated, on account of the red colour of the blood in the larger *Planorbis*. This reaction is certainly due to the gland secretion, the inoculation of which must soften the tissue and help the miracidium in penetrating. The snails are generally indolent; when attacked by a larger number of miracidia, they try to retract the exposed parts or to leave the water.

This first reaction vanishes in a few hours, leaving at most one or more small nodules.

Miracidia which find no occasion to penetrateyin a snail, sink after some time to the ground and die, having first expelled the contents of the glands.

Development of sporocysts of first and second generation.

(Figs. 18—50)

When an antenna of *Planorbis olivaceus* is examined shortly after the infection, the miracidium is seen stretched lengthwise under the skin; the intestine and the glands are indistinct and seem empty but the vibrating lamellae and the cilia of the external epithelium continue to play. Contractions and displacement of the miracidium are also perceived. Finally everything becomes quiet. The epithelium is cast off and there remains only an oval body with thin membrane, in which the germinative cells multiply and become more distinct.

(An antenna, cut off two days after the penetration of the *miracidium,* appeared oedematous. Immersed in physiological saltsolution and compressed by a coverglass, it became sufficiently transparent to permit the examination by an oil immersion lens. The sporocyst was still extended lengthwise, but on its outer side there was a clear space in which the rests of the ciliated membrane were seen. The cilia themselves had already disappeared leaving only indistinct rests. The outline was now very fine and the two poles were equal, as the papilla, the gland-cells withs their ducts and the great vesicular cells had disappeared. In the interior small granules, round bodies of nuclear aspect, vesicular elements and a few hyaline cells were seen in a protoplasmic mass without distinct structure. The four tubular orifices, with their ciliated membrane in lively movement, were still visible and continued so for at least five hours: also the ciliated epithelium of the antenna was well preserved, but after ten hours every movement had ceased; the epithelial cells of the antenna were detached and their cilia had dropped or disappeared.)

After three or four days the secondary swelling of the antennae indicates the evolution of the sporocysts. These simple or moniliform swellings increase in the first forthnight. About twenty days after the infection they become pale and shrunken without disappearing altogether; therefore the infected snails may be recognized even after a long time. But it must not be forgotten that similar lesions may be due to the parasitism of other trematodes.

After several days the distended membrane of the sporocysts has become so thin that it is hardly seen; nor may it be stained in the flattened antenna. Crushing one of the swellings 5-6 days after the infection, one sees a large number of round masses, formed by small cellules with a central body ressembling a nucleus. Some of them occupy the swollen part of the antennae, while others escape by the rupture. Examination in colored sections offers no great advantage.

A few days afterwards, the masses stretch, forming short secondary sporocysts that move about in the lymph of the antennae and for a short time in physiological solution. Constrictions are frequently seen, and seem to indicate a multiplication by transversal division. In water, they perish rapidly. In this period the emigration of the sporocysts towards the visceral sac begins. They are found there 20 days after the infection, forming very long and branched sporocysts containing oval masses, destined to the formation of new sporocysts or cercarias. In these the body is formed first, then appears the stem and finally the branches of the tail. The whole body is still covered by an epithelium with easily stained nuclei. Living and active cercariae appear only after a month and a few days. In other cases, due probably to less favourable temperatures, new sporocysts are apparently formed.

Cercariae with forked tail.

For some time past cercariae with forked tail have been known, though further evolution was ignored. They were considered as curiosites of no apparent importance. They are rarer in Europe than in warmer regions, where they constitute half of the easily found species. In Brazil, I know of four quite distinct species found in different *Planorbis*

(Figs. 65–68) and of one observed in a *Semisinus*.

In 1913 MIYAIRI and SUZUKI published an important work in which they described the evolution of the *Schistosomum japonicum*. The cercaria was found in a fresh-water mollusk with a shell like those of the genus *Bithynia*. It was considered new and called *Blanfordia* or *Katayama nosophora* ROBSON. The description and the illustrations show that the cercaria has six large unicellular glands in the posterior part of the body. According to the authors they are formed in secondary rediae which, however, are really sporocysts. The percutaneous infection was proved by experiments on animals.

The observations of the Japanese investigators were confirmed and amplified by independent studies, made by LEIPER. The latter, as chief of a scientific commission, continued his studies in Egypt where former students, though observing many cercariae, had failed to recognise those belonging to *Schistosomum*. He found four cercariae with corresponding characteristics and was able to obtain adult schistosoms in animals, as well by cutaneous infection as by ingestion of cercariae. As hosts of *Schistosomum haematobium*, he indicated snails akin to *Planorbis* and *Physa*.

Later on he concorded with the distinction between *Sch. haematobium* and *Mansoni* giving *Physopsis africana* as principal intermediary host of the first and *Planorbis Boissyi* as that of the second.

Description of the cercariae of Schistosomum Mansoni.

(Figs. 48 & 51—63.)

The newly formed cercariae, found in great numbers in the viscera of infected snails, are covered, body and tail, by an epithelium with large and closely disposed nuclei, also revesting the suckers and making them stand out clearly. After this has been shed by a kind of moult, tke body becomes more transparent without showing its structure more clearly (Fig. 62).

Fresh preparations were fixed in osmic acid, others in formol and coloured by various methods; segments of infected snails were stained with hematoxylin only, or followed by eosin or VAN GIESON; thus I was able to verify the following details of structure which are not easily seen by using one method only.

The body of the moving *cercaria* shows the varied aspects, observed in other flukes and due to their extreme contractibility. In repose they assume the form of an egg with one end truncate. The greatest length is 0,14, the greatest width about 0,04 – 0,05 mm. The tail shows a very contractile basal part, forming in repose an elongated cone; fixed to its truncate apex is a spindleshaped transverse piece which can be folded in the middle, so as to form a prolongation of the basal part (fig. 60). This has a length of about 0,27 and a (greatest) width of 0,03, each half of the transversal piece being about 0,08 mm. long. Thus the whole length may attain nearly 0,5 mm. With good light, these dimensions are sufficient to show the stationary cercaria in the shape of a short white stroke. When swimming it appears in the very caracteristic form of an 8, due to the rapidity of its movement.

The whole body, tail included, is transparent, slightly opalescent and covered with very minute spines.

The body representing the earliest form of *Schistosomum* has two suckers, the oral one, with a diameter of 0,03 mm., being far larger than the posterior. Its shape is that of an ovoid with the anterior fourth cut off. Its musculary system is insignificant but the mouth and pharynx, situated at the posterior end may be drawn in the cavity and even reach the anterior opening. The movements of these parts are like those of the piston of a syringe and very striking.

Just inside the exterior opening of the sucker there is a ring of 6 or more small spines which, together with the supporting part, may be everted and form a short *rostrum*. Near their base open the excretory ducts of the abdominal glands, the secretion of

which may thus be inoculated in the tissues of the host. There are three ducts on each side, arranged like the strings of a cord (fig. 63). They perforate the anterior sucker and are so wide and sinuous that the abdominal part might be mistaken for a coecum. The ducts are connected with six or more relatively enormous cells with granulated protoplasm, situated partly behind the ventral sucker and occupying a large space, characterized by transparency. They have a diameter of 0,03 mm, while that of the large nuclei is 0,01. The abdominal sucker with a diameter of 0,18 mm. looks very small; it is much nearer to the posterior end and may be retracted or protruded. Coloured preparations show behind the sucker an agglomeration of small and mostly elongate nuclei, other ones between the oral sucker and the large glandular cells and some round the suckers. The intestine is not easily distinguished; it seems to form a simple and short bag after the slightly visible, bulbless pharynx and oesophagus. Outlines of the genital system (which ought to be different according to sex) are hardly recognized. The numerous nuclei, seen in the cercariae, seem mostly connected with the muscular system; the others may belong to the glandular ducts, the nervous system, the intestine or the rudiments of the genital organs, but they are so little differentiated that in spite of much work I could not obtain a clear insight. The tail and its transverse piece are formed by nucleate cells of a certain size. It moves independently, even some time after it has been separated from the body, to which it is loosely attached by a kind of articulation.

After penetration the *schistosomulum* (fig 69) shows no more large glandular cell; they seem to vanish after having fulfilled their end. The ducts also seem to undergo involution; for the rest we find the same structures.

In this species the forked part consists of a separate spindleshaped piece at the truncate end of the tail. It may be bent in the middle so as to lie in the same axis or horizontally extended. This position is assumed while floating at the surface and supporting the body of the cercaria which hangs down. The extremities may also curl inwards (fig. 55). The comparatively small body and the rapid movement give to the cercariae, examined under low power, a certain likeness with spermatozoa.

Examination of infected snails show the cercariae formed in sporocysts, of which large numbers are found in in the liver and the genital gland. In the infected liverfollicles the glandular epithelium disappears; this explains the state of apathy and atrophy, so often seen in the infected snails. Of the infected *Planorbis*, the specimen, which lasted the longest, died after at three months, during which it furnished thousands of living *cercariae*. The production was diminishing towards the end of this period.

Conditions under which the Cercariae leave the snails.

In the complete absence of light, the ripe cercariae do not seem disposed to leave their host, even at temperatures of 30 to 31 degrees, though they may accumulate in great quantities in the mucus expelled by snails. In the refrigerator, in intense cold and complete darkness, the free cercariae remain at the bottom of the water attached by the oral sucker; under these conditions they may live more than 24 and less than 48 hours. When the snail retreats into the shell, as it does before death, the cercariae do not come out, so that, after the crushing of a shell, hundreds of live cercariae may pass into the water which was before absolutely free of them.

Exposing the snails to the sun's rays for several hours, we obtain a great number of cercariae, so that experimental infections are preferably made between three and four o'clock in the afternoon. Bathing at this time must therefore be considered as more dangerous than in the early morning. Light alone, without corresponding heat, is not favorable to the coming out of the cercariae.

When the cercariae are active, they come to the surface of the water where they remain a long time hanging by the transversal part of the tail. When the water is shaken, they move rapidly, the body appearing in the form of the cipher 8.

In a capillary tube, placed on ice for a quarter of an hour, they retain life and movement; but they die in a few minutes in freezing water.

From three small snails that did not furnish any cercariae, an enormous number issued after the hosts had been exposed to rays of a NERNST lamp (without blue glass), until the right water temperature was reached. The diffused light of the mourning hours produced no effect.

Under favourable conditions the first free *cercariae* may be seen after little more than thirty days. At lower temperatures they appear later, which is probably due to the fact that the sporocysts form new ones in stead of cercariae.

In the beginning only a limited number of cercariae issues from the snails and spreads in the water and may easily remain unnoticed. As time goes on, they become more abundant and the swarming out may continue for some weeks. It is not easy to watch them in the act of emerging. I saw them several times issuing from the intestine, but this may not be the only way.

Penetration of the Cercariae.

Sch. cercariae may reach their next stage in man or mammalia by penetrating through their mucous membranes while they are drinking or through their skin while they are bathing or staying in the water for some other purpose. Even the contact with hands or feet may be sufficient. The penetration is easy but its observation is difficult, even in experiments. However, it is clearly proved by the disappearance of the bodies of the cercariae from the water which was in contact, their presence in sections of the skin, reaction on the point where they penetrated and finally the appearance of adult blood flukes after a month or more.

LEIPER observed the infection of rodents and monkeys. In my experiments guinea pigs and rabbits gave good results. The cercariae seem to attack all the mammalia, exposed to them, but in many species the evolution does not reach the adult stage.

In experiments, partial immersion may be used or the animals may be gently but securely immobilized and part of the bare or shaved skin bathed by means of a wide glass tube containing the cercariae. After half an hour the water is full of tails which the cercariae cast off when they pierce the skin. The animals, principally white rats, show signs of irritation, followed for several days by local reaction.

When a piece of skin is removed twenty to sixty minutes after the bath the body of the cercariee may be found in sections. I obtained positive results in guinea pigs, rats and in a small pig which furnished the coloured preparation shown in fig. 69. The *Schistosomulum* is seen in the *rete Malpighii* the head touching the cutis. The glands are empty, as may be found even in specimens which just began penetrating.

The perforation of the mucosae ought to be much easier, but even the horny layer of the outer skin yields to the combined influence of glandular secretion and mechanical action. The cercariae do not choose the hair follicles or glandular orifices, as LEIPER already concluded from an experiment. made on a new born mouse. Here the penetration took place in ten minutes. However my experiments lead me to consider this time insuficient to warrant the perforation of the skin in larger animals.

Evolution of Schistosomum in Mammalia.

The penetration is followed by a period in which the evolution of *Schistosomum* is little known. They probably soon reach the blood-vessels which carry them to different organs; after a certain time they settle down in the portal system.

I obtained positive results in rodents, when many cercariae were used and some

weeks allowed for the trematodes to attain a size which makes them easy to see. Without these conditions the research may give a negative result.

In rodents the development of a considerable number of *Sch. M.* is quite compatible with all the signs ot good health. The worms are evidently well adapted to life in the blood-vessels and their consumption of blood is not sufficient to cause serious anemia. The symptoms are mostly due to the laying of the eggs and their migration which only begin after some weeks.

In the excrements of infected guinea-pigs eggs were not found till after a period of 2 and 1/2 months and then only in small numbers. In the worst infection (in a rabbit) eggs were rare in the sub-mucosa and there were none voided; it may be that in man the conditions are different but, in the cases of rabbits and guinea-pigs, we may conclude that the elimination of eggs is a slow process and that it generally happens some time after the parasites have reached maturity.

Some observations in human pathology and some of the experiments of LEIPER seem to indicate that the elimination of eggs took place rather sooner, five or six weeks after infection. I think the fresh cases must have been very heavity infected and that later on the eggs would have been more numerous.

After three weeks adult worms are found in the mesenteric veins which are generally very congested. The veins of the liver contain about the same number. After five weeks the sexual organs are well developed; in the uterus the females show a ripe egg. The dimensions of the flukes vary considerably, according to the degree of muscular contraction. The specimens which die slowly in the tissues of the host, are quite relaxed. But even so the dimensions are slightly inferior to those given in the old literature. I do not think that this is due to the difference of the hosts, as the worms, found in autopsies of infected people, were not distinctly larger, but perhaps the species *Mansoni* may be smaller than the *haematobium* of which the females contain many eggs, instead of one only. The intestine of the female can be easily seen, as it is full of black matter, formed from the absorbed and digested blood. The same may be said of the male though in a lesser degree. In sections of the liver, the females, cut transversally, would be little conspicous, if they were not characterised by the intestinal lumen showing in one or two places, according to the region. Besides this, the sections of the excretory canals are seen. The male is more characteristic, due to its form and to the spines on its body.

Symptomatology of Schistosomatosis.

There are several methods for determining the symptoms of infection by *Sch. Mansoni.* We can study men (naturally) and animals (experimentally) infected with the disease, verifying the objective symptoms and taking note of the complaints of the patients; or we may begin with the macro-and microscopic anatomic alterations. Whe have another resource in the comparative study of the lesions, produced by other species of *Schistosomum* in men and domestic animals. The most important is the *Schistosomum Japonicum* producing very intense infections, as well in man, as in the larger domestic animals. They are generally located in the same organs and have been widely studied 9).

The first observations on *Schistosomum* infections, made in the Old World, may be used but only with great caution, as the symptoms belonging to two species of *Sch.* and other intestinal parasites are frequent in the same place.

The presence of adult worms in the veins is mostly well borne (which agrees with the general rules of parasitism); it is proved by the fact that a rabbit may have half a thousand worms in the mesenteric veins without showing any particular symptoms.

On the other hand, LETULLE attributes the phlebitic processes, observed in cases of long standing, to the parasitism of the adult.

Glands with toxic or irritating secretion are unknown in adult schistosoms, but cer-

tainly the products of their metabolism are poured into the circulatory system of their host 10). They are however diluted and carried along when the circulation is free, as in the larger, veins. The blood, absorbed by the schistosoms, is their only alimentation but it is not so much that in moderate infections, it could not be easily. substituted. For this reason anemia is no help for recognising the infection. People often attribute diarrhoea to the parasitism of worms and some authors, amongst them GONÇALEZ-MARTINEZ, consider it as a symptom of the infection of *Sch. Mansoni*. According to my experience, it is only a frequent complication, as in other worm diseases and in chronic cases one finds habitual constipation which is explained by the sclerosis of the intestine. In recent cases the number of stools is generally not increased.

JESUS-RISQUES called attention to undefined pyrexy of uncertain origin which may be attributed to *Sch. Mansoni* infection 11).

I was present at the autopsy of a patient infected by these parasites who succumbed to a not very characteristic pyrexy. This subject is worth considering. It is already proved that acute and recent infections of *Sch. Japon.* may cause fever which is often acompanied by eruptions somewhat resembling urticaria.

There is another symptom, which in my experience, is more frequent and which may be explained, taking into account the anatomical observations made in patients, infected by *Sch. japonicum* and *M. pulmonalis*.

These patients often complain of gjddiness which I attribute to the embolism of the blood flukes or their eggs.

Some patients, who were accustomed to bathe frequently in ponds, told me; that they felt itching afterwards. Even the name of two of the ponds, we visited, refer to the itching. Though this symptom may not be considered pathognomonic, tt always gives a pretty sure indication of infected water and leads us to expect penetration of cercariae, which causes irritation, as is well seen n the experiments on white rats 12).

There is a series of symptoms, observed only in cases of intense or often repeated infection, which concern the intestine and the liver and which are explained by the anatomical lesions, observed in those organs.

These, however, depend rather on the eggs than on the worms, the eggs being deposited in the tissues, as we shall see in the pathological anatomy. The symptoms of the liver and intestines are, however, not very characteristic and may be found in many other affections; so their value depends on the exclusion of other complications or accidental diseases, observed in infected individuals who, in some places, form the majority of the population. The frequency of certain symptoms might be demonstrated by statistics but in this line nothing has been done in this country 13).

Sanguinolent mucosities may be attributed to *Sch. Mansoni,* if there are many eggs and no amoebae. Icterus, hepato-and splenomegalia, retraction of the liver and ascites are also suspicious symptoms when there is no probability of other etiology.

Eosinophilia is observed in schistosomatosis as in other forms of helminthiasis, without being of much importance for diagnostical purposes, as the greater part of the patients have also other worms. It is rather by an examination of the fecal matter than by one of the blood that a reliable diagnosis may be obtained, excepting only cases of quite recent infection.

The other affected organs do not furnish any clear symptoms. In the first phases of their evolution, the worms probably pass through the pulmonary circulation; some not very characteristic manifestations, such as bronchitis and cough, may be due to them. The pancreas is often affected; it is advisable therefore to seek for indications of glycosuria, which has not yet been done systematically.

Besides the attacks of giddiness, nervous perturbations are observed which may be due to schistosomatosis; these are lapse of memory, want of energy, feeble comprehen-

sion and reasoning, periods of apathy and bad temper, etc.

Complications and secondary affections.

Infection with *Sch. M.* is so common that the coincidence of other pathological conditions must necessarily be very frequent. Thus liver abcesses may be due to *amoebiasis* and may be attributed to the schistosoms, simply because their eggs are found. *Appendicitis* of other origin may take place in an appendix, infiltrated with eggs. Even symptoms which are generally observed in *Schistosomum*-infection may be partly due to complicating conditions, as tuberculosis, syphilis, alcoholism and heart diseases. In severe affections of the rectnm (which seem particularly frequent in Egypt) the *Sch. haematobium* may be partly responsible. Haemorrhoids may be altogether independent or a consequence of *cirrhosis hepatis.* Anal fistule may be independent or due to mixed infection. (I dont know of any attributable to *Sch. M.* alone). Adenoms and papilloms, found from the ileum to the rectum in increasing number, may be due to intense and long standing infections but the malignant degeneration of them ought to be considered a complication. *Haemoptysis*, observed in chronic cases, may be dependent on other causes.

On the other hand the parasitism of *Sch. M.* may favour complications and secondary affections. It certainly proves the existence of sanitary conditions which favour an infection, not only by other worms but also by protozoa and bacteria.

Pathological anatomy.

The results of the parasitism of *Sch. M.* are better unterstod by macro-and microscopical investigation of the anatomo—pathological conditions than by the symptoms, observed in life. These results allow us to approach certain questions which are still somewhat obscure. I shall first state my personal observations made on animals experimentally infected.

In rabbits, guinea pigs and white rats, the presence of the blood flukes is hard to prove during the first period after experimental infection, evidently, because they have not yet reached their final habitat. This agrees with the results of other helminthological experiments. The youngest and smallest specimens were found by LEIPER in the liver of white rats.

After three weeks, we may count with positive results. On opening the animal, the mesenterial veins are full of blood and the tissues are so transparent that the males are easiey seen, their white colour being in contrast with the surrounding blood. They are found in all the larger branches, from the stomach to the end of the intestine, without any preference for the last part. By dilaceration of the liver in physiological salt solution, a similar number may be obtained. The females are found in about equal numbers with the males occupying their *canalis gynaecophorus* but after some time the sexes separate.

In recent infections, the veins, although full of flukes, show no distinct lesions. The neighouring tissues, the liver and the intestine are free from macroscopic alterations.

Complete development and egg production require one or two weeks more. Even in heavy infections the eggs do not appear immediately in the excrements which shows that their emigration is slow.

After many months the larger veins may be found empty, but sections of the liver and the intestine show some males and many females, isolated in the smaller veins.

In the liver the females are found in the small interlobular veins, more rarely in dilated capillaries or small arteries, never in bileducts or in the glandular tissue. The bloods vessels seem very congested but there is no inflammatory reaction around them. In the inicial time, eggs in liver preparations are rarer than sections of flukes but they are often surrounded by an infiltration of round cells. In severe infections of older standing the whole interstitial tissue may be thus infiltrated, which may be considered as the

first stage of a cirrhotic process. The capillaries seem much distended.

In the intestine I found the males near to the serosa; the females can reach the submucosa. The eggs are found in small groups in the mucosa at the base of the glands and also isolated between them, but sparsely and without indicating, how they reach the surface. The groups of eggs are surrounded by an infiltration not unlike a tubercle. Giant cells are formed exclusively arround or inside of the empry shells. Eosinophilous cells are seen in the tissue and in the vessels. In older infections the eggs may also abonnd in the submucosa.

On the lesions observed in man much has been written. The first writers did not distinguish between the two infections found in Egypt and most of the older or newer authours treat in preference of fatal cases where the infection was many years old. In such cases, in the sections the flukes may be rare or even absent. The predominant lesions are due to the eggs which exist in large, often enormous numbers. Small lesions without great importance, when constantly repeated, lead finally to severe and extensive lesions of the most invaded viscera.

Material from early stages is rare. However, I have elements for describing them, using personal observations and others found in the litterature.

Of macroscopical lesions the only ones, observed in more recent and not very severe infections, were a swollen spleen and medullary tumefaccion of the mesenteric lymph nodules, both very common in other diseases. The flukes are not seen in the mesenteric veins and the best places to look for them are the trunk and the ramifications of the portal vein.

In older and more severe infections there are lesions of the liver which may look either like hypertrophic or like atrophic cirrhosis and may cause ascites and jaundice 14). The spleen which as a rule is free from flukes and eggs may be swollen or small. Of other organs only the intestine shows striking alterations. They may consist in a varying degree of general sclerosis and alterations of the mucosa, more apparent in the rectum and diminishing in proportion of the distance from it. The difference in localisation between man aud animals may be due mostly to gravitation.

The histological alterations of the organs were studied in my material and also in preparations received from Dr. J. RISQUES in Venezuela and compared with good preparations oi infections with *Sehistosomum japonicum.* Some were from human infection, made by Prof. CROWELL in the Philippines and others from domestic animals, received from Prof. AKANASCY. The infections were severe and the corresponding lesions were well accused.

Individuals, who suffer from infection with *Sch. M.* beginning in early youth, may show few flukes in comparison with the enormous number of eggs, accumulated in the tissues, without the slightest probability of leaving them during the life of the host. They are more numerous in the liver and the wall of the intestine, but are found also, though in smaller number, in other organs, as the mesentery and its lymph glands, the pancreas, the lungs and the brain. Like the eggs of *Sch. japon.* they are often found in the interstitial tissue of the viscera, independent of any celular infiltration; they may be more or less calcified which points to their staying there for a long time. Such deposits are the cause of the sclerotic process, found principally in the terminal part of the gut and in the liver, where they lead to a form of cirrhosis, with or without splenomegaly, jaundice and ascites.

The intestinal sclerosis impares the funtion and leads to obstipation. Though most pronunced in the rectum, it extends upwards and there are many accounts of thickening of the appendix, due to infiltration with eggs. It may even extend to the higher parts, though it does not call the attention of the observer.

Macro— and microscopic lesions of the intestine where studied accurately by LE-

TULLE in a case from Martinique, infected most likely by *Sch. M.* only, though the author speaks in somewhat vague terms of the coexistence of eggs with terminal spine. His descriptions agree with those of the authors who observed very severe cases and some of them may be found also in less intense infections. Similar lesions may also appear in *Sch. japon.* infections which are frequently severe.

LETULLE describes superficial ulcerations of the mucosa, different from those dependent on amoebiasis. In other places he noted a proliferation leading to the formation of polypoid pedunculated growths with the caracter of adenoms. They might be traversed by a fibrous axis and have an ulcerated surface.

LIEBERKUEHN'S glands may be destroyed by ulceration, atrophy or cystic dilatation, followed, suppuration, or become hypertrophic or hyperplastic. The epithelium shows the corresponding alterations without undergoing a typical proliferation.

The interstitial tissue of the mucosa also shows signs of, sometimes extreme, hypertrophy The capillaries may appear numerous and distended, while the interstitial tissue may force the glands assunder and spread on the surface of the mucosa, below the epithelium when it still exists. Interstitial hemorrhages and pigment deposits were never seen.

The muscularis mucosae is not reached by the ulceration nor does it show other alterations than hyperplasia.

The submucosa is transformed into sclerotic tissue, while the adipose cells disappear.

The layers of the muscularis propria only show general hypertrophy, real or apparent.

The serosa was also sclerotic in LETULLE's case, but the products of parasitism. observed in other cases, were not seen.

The lymph vessels show no alterations, except some cellular infiltration near to the foci of inflammation and ulceration. They are not used for the migration of the worms and eggs. The lymph follicles are not invaded by the parasitical process. Also the nerves and ganglia show no lesions.

Except in the parts which pass through the muscular layers, the veins undergo a special and characteristic process of hypertrophic and sometimes obliterating endophlebitis. The remaining free space is generally excentric, the process being limited to part of the intima. It may be seen alo on the mesenteric vessels and in the veins of the pelvis, which do not properly belong to the portal circulation. A hyperplasia of the media is also noted here.

Oviposition and migration of the eggs.

The main questions are: How do the eggs reach the extravascular tissue and how do they arrive in the intestinal contents? The solution of these questions is very difficult but seems to have hardly troubled most of the authors. Following LETULLE, they took for granted that the females advance in the small veins until they plug the narrower vessel, and then depose their eggs in them. They think that the increased pressure, following the plugging of the veins, forces them through the wall, and that afterwards they make their way to the diffent places were they are found. As for the eggs found in the lungs, in the brains etc. they were supposed to be carried by the bloodstream.

What may be the force which makes the eggs, relatively large and motionless bodies, advance in the tissues? Here the authors evidently thought of the eggs with terminal spines, which might help the progress in one direction while the other blunt end prevents a retrograde movement. But the lateral spine would not help the advancing, while it might oppose any motion in which the spineless end does not go first. And the eggs of the *Sch. Japonicum* have not even got a spine. It is true that a diminutive thorn has been described, but it is generally not terminal and is often missed altogether, as well in preparations from stools, as in preparations from tissues. It seems far from constant. Also the point is often curved and generally not very sharp.

Nobody seems to have considered, that it would be much easier for the worm to

perforate the wall of the vein and pass through it, if not with the whole body, at least with the anterior part, also the position of the genital opening and the analogy with other worms suggest this idea. The very endophlebitis speeks for it, as it is always excentric (even when almost obliterating) and thus indicates a localized lesion.

It is true that this process of oviposition has not been witnessed in the preparations, as far as the literature and my experience go, but neither has the oviposition in the vein.

It is easier to unterstand how the eggs, which are so often seen at the base of the mucosa, may come to the surface, though the bloodstream in the veins and the lymphcirculation go in the opposite direction. They generally follow the tissue between the glands of LIEBERKUEHN and do not pass through them (as the preparations clearly show). The only force acting in this direction is the reconstitution of the tissue. No abscess formation helps the elimination and the ulcerations, when present, are quite superficial. I cannot share the opinion of LETULLE, who thinks that the dilated and sometimes suppurating glands, help the escape of eggs and miracidia, nor can I admit that all the living eggs should have passed through the warty or pedunculated adenoms which are found in old cases. They are more likely to be the source of the degenerated and calcified eggs which are abundantly found in the stools of certain cases.

As for the eggs, seen in the submucosa and under the serosa of the intesiine, in the liver, pancreas, brains and lungs, they mostly fail to reach the surface in a living state, as long as the host is alive. They accumulate in the tissues, where they are seen in great numbers, and, after provoking during a short period an inflammatory reaction, they finally are incapsulated in a cicatricial tissue without a sign of reaction. Calcification sets in after an indeterminate, but probably very long, period and may indicate the final death of the egg.

Living eggs are not known to have been found in the bile or pancreatic secretion. I examined the contents of the gallbladder several times with constantly negative results. In sections the eggs are never seen in the bile ducts.

Other worms are known to deposit their eggs in the tissues of their hosts, for instance the *Hepaticola hepatica*, the eggs of which are very common in the liver of the common rat and have often been taken for coccidia. In this case it is quite sure that they are deposited by the adult worm which afterwards dies in the same place. The eggs do not reach the outside world, unless the host is eaten by any animal or dies in some other way.

Besides living eggs in their earlier stages and calcified ones, the tissue may contain empty shells, as the typical form of the tear and giant cell formation clearly show. They are found in different situations and not only near to LIEBERKUEHN'S glands, as might be supposed from LETULLE'S description, who considers the escaping of the miracidium by the hollow space of the gland as a normal way of spreading the infection. We may state that neither he nor anybody else ever saw a free embryo in the sections. In my opinion it is extremely unlikely that miracidia, escaping from the eggs in the tissues, should pass through the intestine alive and, even so, they would die soon, unless they came immediately in contact with water. I cannot believe that the rather rare ocurrence of ecdysis in the tissues is of any advantage for the propagation of the flukes, but the fact is very interesting and hard to explain.

Undoubtedly in the infection with schistosoms as well, as with many other worms, there is an enormous waste of eggs which in our case is not even compensated by a large production. The real compensation is given by the enormous multiplication in the organisms of the infected snail.

In the larger number of cases the ripe eggs appear mixed and in close contact with the common elements of the stools and not involved in bloody mucus. This constant

output not only insures the conservation of the species, but indicates the existence of a relatively quick and easy way of leading the eggs to the outside world without striking symptoms. Thus the facility, with which the infection is overlooked, may be understood.

Prognosis, Therapeutics and Prophylaxis of Schistosomatosis.

From our observations already refered to, one may conclude that in our country schistosomatosis is mostly not severe. GONZALEZ-MARTINEZ estimates that 45 % of the infected show no clear symptoms and I think that in Brazil the proportion of unremarked cases is rather higher than lower. On the other hand I dont know of any treatment for curing or improving this infection 15), while some of my observations show that these flukes may live for many years in the human body. Therefore our principal efforts must be directed to the prevention, rather than to the cure of this infection.

Schistosomatosis is always dependent on water, either used for drinking or coming in contact with the skin. As its infectiveness is necessarily connected with the presence of certain species of Planorbis, it is evident that prophylaxis ought to be directed in the first line against these intermediary hosts. The water collections might be cleaned by removing mud and aquatic vegetation and catching the specimens in sight, but these measures will be often difficult or impossible just there where they are most needed.

There are other measures for preventing the infection of the snails, which would also tend to eradicate the even more common and dangerous *ankylostomum* and *necator* infections. They are directed against the contamination of soil and water by human excrements. It is clear that sewers opening into rivers, the water of which is used for bathing and washing, could not be tolerated.

If suspect water must be used, a storage for 24-48 hours would be a sufficient protection. Early in the morning the water is not likely to contain cercariae; taken near to the surface and stored for a few hours it will lose any power of infection. The same may be achieved by heating (which need not attain the boiling point) or by desinfection. According to LEIPER it is sufficient to heat the water up to 50 % or add one gramme of sodium bisulfite to the liter.

The danger of bathing in stagnant water.

Planorbis (of the larger species) are found in waters with little or no current, not subject to complete evaporation and preferably with floating or rooted water plants Such waters may be dangerous, when drunk, but would be avoided, when there is a choice in the supply of drinking water. But already the contact with the bare skin of legs and arms, as in fishing and washing, may cause more or less severe infection; however the greatest danger consists in total and prolongated immersion while bathing, as the chances of infection increase in proportion to the surface exposed and the time of contact. I know the history of several patients who were in the habit of bathing in stagnant water with water plants and *ampullariae.* These much larger snails are eatable and better known; they may thus serve as indicators for suspect water. A few patients had even noticed the presence of *Planorbis.* In some places, for instance in Aracajú, *Planorbis olivaceus* may become so abundant in dry times that it is used for feeding pigs. There and also in Laranjeira, we found ponds, whose popular name indicates that people who bathe feel itching afterwards. This is quite a caracteristic symptom of the penetration of the cercariae, as experiments in animals, principaliy in white rats, clearly show.

To become infectious the water collections must be contaminated with *Sch.* eggs coming from the intestine of man or animals. This may happen at any time but mostly in consequence of showers and inundations. Of course the water must also contain specimens of *Planorbis.* At a sufficiently high temperature, after 4—5 weeks, there may be infected

snails in condition to furnish living cercariae for two or three months. Thus a contamination, taking place every second month, would be quite sufficient to make the water permanently suspect.

We know by experience that the cercariae dont issue at any time. It is next to impossible to find a cercaria early in the morning, though the water may contain infected snails, while in the afternoon, after several hours insolation, they are quite abundant. Therefore a short bath in the morning may prove harmless, while a protracted bath during or after the warmest hours ma leady to a multiple infection. The dry season, when there is more sunshine and less water, while its temperature is higher, must be the time when most of the infections are acquired.

Litterature:

The litterature on the blood flukes and the pathological conditions caused by them is very large; LEIPER's report contains an alphabetical list of 562 numbers. A few more may be found in other papers cited. I give a list of papers which refer specially to my subject and principally of those which have been studied and quoted. I have not enumerated the numerous treatises on helminthology, tropical diseases or general pathology and medicine, as they are generally well known or contain nothing new.

The litterature on *Sch. jap.* is given more extensively, as the subject is new and intimately connected with mine. Not only do *Sch. M.* and *Jap.* provoke similar symptoms and lesions but those of the latter are generally better known and more easily studied, the infections as a rule being less complicated, more acute and more intense. Therefore their study is very useful to the student of *Sch. M.*

Schistosomiasis in man, observed in Africa or of African origin.

SIEBOLD, C., 1852 – Ein Beitrag zur Helminthographia humana. Aus briefl. Mitth. d. Dr. Bilharz. – Zeitschr. f. wiss. Zool., H. 1, pg. 53, Bd. 4.

BILHARZ TH., 1853 – Fernere Mittheilungen ueber Distomum haematobium. – Ibidem, H. 4, pg. 454.

KARTULIS., 1885 – Ueber das Vorkommen der Eier des D. haematobium Bilharz in den Unterleibsorganen. – Virchows Arch. Bd. 99, pg. 139.

LEUCKART R., 1886 – Die Parasiten des Menschen etc. Zweite Aufl., Ad. 1, Abth 2.

HILL, B., 1888 – Clinic. lect. on haematuria. – Brit. med. Journ., V. 1, pg. 199.

FRITSCH., 1888 – Zur Anatomie der B. haem. – Ztschr. f. mikr. Anatomie, Bd. 31, pg. 192.

BROCK, G, S., 1893 – On the anat. and physiol. of the ovum of Bilh. haemat.-Lancet, Sept. 1893, pg. 622 – 625.

SONSINO P., 1893 – B. haem. and B. Disease. – Davidson, Hyg. a. Dis. of w. Climates, pg. 90.

BROOKS, 1897 – A case of D. haemat. – Medical Record 1897, pg. 492.

GOEBEL, 1903 – Clin. a. pathol. Observ. on B. Disease. – Journ. of trop. Med., pg. 106.

FREEMAN., 1905 – Journ. of the R. Army med. Corps, Vol. V, pg. 145 (278).

GOEBEL, 1909 – Die patholog. Anathomie der Bilh. – krank. – Berl. klin. Wchnschr., pg. 107.

RUFFER M. A., 1910 – Note on the presence of Bilharzia haematobia in Egyptian mummies of the 20th dynasty (1220 – 1000 B. C.). – Brit. Med. Journ. Vol. 1 pg. 16.

JOYEUX, 1912 – Notes sur quelques cas de Bilharziose, obs. à Kouroussa, Guinée fr.-Bull. de la Soc. de Path. Ex. Vol V.

BOUR E. F., 1912 – On num. cases.... with a contr. to the study of Bilharziosis in Mauritius. Journ. of trop. Med., Vol. X n. 10 pg. 148.

ARCHIBALD, R. G., 1914 – Intest. Schistosomiasis in the Sudan. Brit. med. Journ. pg. 297.

ARCHIBALD, R. G., 1914 – Treatment by autogenous vaccines. Trop. Dis. Bull. V. III n. 6, pg. 202; VI pg. 202.

CONOR, A., 1914 – Essai de la transmission de la Bilharziose. – Bull. de la Soc. de Path. Ex. – T.

Arch. de l'Inst. Pasteur de Tunis. T. IX, F. 1.

CHENHALL, W. T., 1915 – B., compl. by adenocarcinoma... Med. J. of Austr., Vol. 2, n. 16, pg. 339.

LEIPER, R. F., 1915 – Report on the results of the B. Mission in Egypt. – Journal of the Royal Army Med. Corps, pg. 2 – 55, 148 – 192, 253 – 267. To be continued.

1916 – Ibidem, Vol. XXVII, pg. 171.

1918 – Ibidem, Vol. XXX, pg. 235.

CAWSTON, F. G., 1915 – Schistosomiasis in Natal. – Journ. of trop. Med., n. 22, Nov. 1915.

CAWSTON, F. G., 1916 – Schistosomiasis in Natal. – South Afric. Med. Rec., Vol. XIV, n. 4, pg. 53.

CAWSTON, F. G., 1917 – The cause and effects of Bilh. Dis. in South Afric. Egypt and the Far East. – South Afr. Med. Rec., Vol. XIV, n. 11, pg. 163.

LAWTON, F. B., 1918 – The early symptonis following infection by *Sch. M.* – J. R. Army Med. C. XXXI, n. 6 pg. 472.

II

Schistosomum haematobium in Asia.

HATCH, Y. K., 1887 – B. haemat. – Lancet, April, pg. 875.

STURROCK, P. S., 1899 – B. in Mesopotamia. – Brit. Med. Journ., pg. 1543.

SEWELL, E. P., 1903 – B. in India. – Ibidem, pg. 490.

SEWELL, E. P., 1904 – Case of B. haem. – Journ. of the R. Arm. Med. Corps, Vol. 2, pg. 346.

SCOTT, J., 1904 – B. haem. in Persia. – Brit. Med. Journ. 1, pg. 725.

WARDROP, D., 1906 – Report on five cases of B. – Journ. of the R. Arm. Med. Corps, V. 7, pg. 282.

HOOTON, A., 1914 – A case of B. Diasease. – Ind. Med. Gaz., pg. 188.

MILTON, F., 1914 – Does B. exist in India ? – Ibidem, No. L. (Trop. Dis. Bull., V. 3, pg. 289).

III

Schistosomum Mansoni in America or of American origin.

MANSON, P., 1902 – Journ. of trop. Med., Dec. 15.

MANSON. P., 1903 – Tropical. Medicine. – Ibidem 1905 e 1907.

GONZALEZ MART., 1904 – La Bilharziosis en Puerto Rico.

LETULLE, M., 1905 – Bilharziose intest. – Arch. de Paras., Vol. 9, pg. 329.

SAMBON, L., 1907 – Journ. of trop. Med. X, pg. 117 & 303. – Idem XI, pg. 31, 1908.

HOLCOMB, R. C., 1907 – The West Indian Bilharziosis in its relat. to Sch. M. – U. S. Naval Med. Bull. Vol. 1, Nº. 2.

PIRAJÁ DA SILVA, M., 1908 – Contribuição para o estudo da Schistosomatose na Bahia. – Brasil Medico, pg. 281, 441 & 415.

PIRAJÁ DA SILVA, M., 1908 – La Schistosomose à Bahia. – Arch. de Paras., Vol. XIII pg. 451.

PIRAJÁ DA SILVA, M., 1909 – Contribution to the study of Schistosomiasis in Bahia. – Journ. of trop. Med., Nº. 11.

LOOSS, A., 1908 – What is "Schistosomum Mansoni" Sambon 1907 ? – Annals of trop. Med. and Parasitology, Vol. II, Nº. 3 and repr.

SAMBON, L., 1909 – What is Schistosoma Mansoni ? – Journ. of trop. Med. pg. 111.

NOC, F., 1910 – La bilharziose à Martinique. – Bull. Soc. Path. exotique, Vol. III, pg. 26. Paris, 1910.

FLU, P. C., 1911 – Beitrag zur Loesung der Frage, ob Sch. M. identisch ist mit Sch. haem. – Centralb. f. Bakt., Abt. I, Bd. 61, pg. 389.

GONZALEZ MART., 1916 – Invest. on the preval. and clin. feat. of Int. B. (Sch. M.) in Porto Rico. N. Orl. Med. Journ. Vol. 9, Nº. 5.

LEIPER, R. F., 1916 – On the relation between the terminal-spined and lateral-spined eggs of Bilharzia. – Brit. Med. Journ., Nº. 2881, pg. 411.

RISQUEZ, J. R., 1916 – Apuntes sobre la Bilharziosis em Venezuela. – Caracas.

IDEM, 1917 – Febres prolongadas etc. – Vargas (Caracas), Nº. 6.

IDEM, 1917 – B. hepatica com ictericia. – Ibidem Nº. 7.

IDEM, 1917 – Docum. p. el diagn. clinico de la B. – Ibidem Nº. 9.

IDEM, 1917 – B. apendicular. – Ibidem Nº. 12.

IDEM, 1917 – Observ. ac. de la dissem. de los huevos etc. – Ibidem Nº 14.

IDEM, 1917 – Nota prel. de la B. puls. Ibidem Nº. 15.

IDEM, 1917 – Nota s. la coex. de L. lesiones pleuro-pulm. etc. – Ibidem Nº. 17.

HURTADO, B. P., 1917 – Hepat. produc. p. el Sch. M. – Ibidem Nº 9.

IDEM, 1917 – Hepatitis produc. p. el Sch. M. – Ibidem Nº. 17.

ITURBE JUAN, 1917 – Distr. y prof. de la B. em Caracas. – Ibidem Nº. 6.

IDEM & GONZALEZ., 1917 – El huesped int. del Sch. M. – Ibidem Nº. 8.

POU R. PINO, 1917 – Form. clin. de la B. – Ibidem Nº. 12.

LEGER, M., 1917 – Schistosomum mansoni Sambon à la Guyane Française – Bull de la Soc. de Path. Exot. T. X, Nº. 6 pg. 464.

ALMEIDA, ELP. de., 1919 – Contrib. ao est. da Schistosomose Mansonica. – These. – Rio de Janeiro, 1917.

CORT, W. W., 1919 – Notes on the eggs and miracidia of the human Schistosomes – U. of California Publ. in Zool. Vol. 18, Nº. 18, pg. 509 – 519. (Jan. 4th.)

IV

Schistosomum japonicum.

KATSURADA, 1904 – Annotat. Zool. Japan., Vol. V, pg. III, 1904.

CATTO, J., 1905 – Sch. Cattoi, a new blood fluke of man. – Br. Med. Journ. N°, 2.

LOOSS, 1905 – Sch. japon. KATS. – Centralbl. f, Bakt., Origin., XXXIX, p. 280.

SCHEUBE., 1905 – Ein neues Sch. b. Menschen. – Arch. f. Schiffs – u. Trop. – Hyg. IX, p. 150.

BEVER, 1905 – Am. Med., X, p. 575.

STILES, 1905 – Ibidem, LX, p. 821,

WOOLLEY, 1906 – The ocurrence of Sch. jap. in the Philippines. – Phil. Journ. of Sc., I, N°. 1.

TSUCHIYA. 1908 – Ueb. e. neue par. Krankh. (Sch. jap.) – Virch. Arch. CXCIII p. 323.

MANSON, P., 1908 – Sch. jap. in a European. – Journ. of Trop. Med., Nov. 16.

PEAKE, E. C., 1909 – Three cases of infect. by Sch. jap. – Ibidem, March.

KATSURADA., 1910 – Bemerkungen z. Lebensgesch. d. Sch. jap. – Centralbl. f. Bakt., Origin., LIII, Febr.

LAMBERT, 1910 – Transact. of Soc. of trop. Med. and Hyg., March and April and March 1911. LIII, p. 519.

HASHEGAWA, J., 1910 – Bemerk. z. Lebensgesch. d. Sch. jap. – Centr. f. Bakt.

SKINNER, 1910 – Inf. by Sch. jap. – Journ. of trop. Med. May 1.

LOGAN, 1911 – China Med. Journ. March. (Sch. j. – dys. in an Am. child).

WILLS, 1911 – Ibidem, March.

LEIPER, 1911 – Note on the pres. of a lat. spine in the eggs of Sch. j. – Transact. of te Soch. of trop. Med. March.

LAMBERT, 1911 – Schistosomiasis and urticarial fever. Ibidem, Oct.

THOMPSON, 1911 – Hankou Reports Annual.... Ed. by Douglas Gray, Brit. Legat. Pekin (2d Ser. for 1911.)

LAMBERT, 1912 – Notes on early stages of an infect. with Sch. j. – Journ. London school of trop. Med. I, N°. 2, p. 145.

BASSETT-SMITH., 1912 – Sch. j. infect. in a Europ. – Trans. Soc. trop. Med. n. 6, p. 219.

YONEJI, MIAGAWA., 1912 – Ueber den Wanderungsweg des Sch. j.... Centralbl. f. Bakt. Abt. I, Bd. LXVI, p. 406.

(IDEM, " 1913 – Ueb. d. Wanderungsweg d. Sch. j. d. Vermittl. d. Lymphgefaessyst. – Ididem, LXVIII, H. 2.

(IDEM) " 1913 – Bezie. zw. Sch. j. u. d. Dhermatitis.... Ibidem, LXIX, H., p. 132.

MIYAIRJ & SUSUKI., 1912—On the developm. of Sch. j. – Tokio Med.

IDEM, 1914 – Idem, D. Zwiscenwirth d. Sch. j. K. – Mitt. a. d. med. Fak. d. Univ. Kyushu Fukuoka, Jap. I, Verl. i. d. Univ.

MYAKAWA Y., 1913 – On the nutrit. of the eggs of Sch. j. – The Sei-I-Kwai Med. Journ. XXXII, N°. 2.

KATSURADA., 1913 – Sch. j. – Centr. F. Bakt. Abth. I, p. 363.

LANING, 1914 – Sch. on the Yangtze river. – U. S. Nav. med. Bull. Jan.

BROAIRD, D. & C. R., 1914 – Sch. j., a clin. & pat. studv of 2 cases. – Am. J. Med. Sch., V. 148, N° 2, p. 187.

LEIPER & ATKINSON., 1915 – Obs. on the spread of As. Sch. with a note on Katayama nosophora by Robson. – Br. Med. Journ., Jan. 30th. N°. 2822.

LEIPER, R. F., 1915 – Report on the results of the B. Mission in Egypt. Journal of the Royal Army Med. Corps, pg. 2–55, 148–192, 253–267. To be continued.
1917 – Ibidem, pg. 171.
1918 – Ibidem, pg. 232.
MANN, W. L., 1917 – Some Pract. Aspects of Sch. as found in the Orient. – Journ. Am. Med. Ass. LXVII, p. 1366 and reprint.
LAWTON, F. B. 1918 – They earl symptoms following infection by Sch. M. – J. R. Anny Med. C. XXXI, Nº 6 pg. 472.
CORT, W. W., 1919 – The cercaria of Sch. japonicum Katsurada. – U. of California Publ. of Zool. Vol. 18 n. 17 pg. 485–607 (January).

V

Schistosomum species in domestic animals.

SONSINO P., 1876 – Int. a un n. par. del Bue. . – Rend. dell. Acc.... d Napoli XV p. 34.
GRASSI & ROVELLI 1888 – La B. in Sicilia. – Rend. d, R. Acc. dei Linc. IV.
SANFELICE & LOI., 1888 – Di alcune inf.... in Sardegna. – Gagliari.
BARBAGALLO, P., 1899 – Contr. allo stud. della B. crassa in Sicilia. Arch. de Paras. 88, p. 277.
MONTGOMERY, R., 1906 – E. – Journ. of trop. Vet. Sc, XV, pg. 147.
VRYBURG, A. 1906 – Centralbl. f. Bakt., Orig., XLIII, p. 806.
LEESE, A. S., 1911 – B. in the Camel. – Journ. of trop. Vet. Sc., VI. p. 263.
SKRJABIN, K., 1913 – Sch. Turkestan., n. sp., ein n. Paras. d. Rindes a. Russisch Turkestan. – Zeitschr. f. Infektionskr. d. Haustiere, XIII, p. 407.
GLEN-LINSTON & SOPORKAT, B. 1918 – B. am. anim. in India. Life-Cycle of Sc. spindal, in Plan. in Bombay. – Ind. Journ. of Med. Res., V, Nº. 4, April.

VI

Schistosomum and allied genera.

ODHNER, T., 1913 – Z. Natuerl. Syst. d. dig. Tremat. – Zoolog. Anz. XLI, p. 54.

VII

List of Litterature.

STILES & HASSALL, 1908 – Index to Catal. of Med. & Vet. Zool. Gov. Print. O., Washington.

Notes.

1. (Pg. 108 after the title.) The helminthological and malacozoological litterature used for my studies belonged to me or to the libraries of our institute and of the *Museu Nacional*. A large part was seen only after the termination of my first studies, begun in the second trimester of 1916 and published in the Brazil Medico in two preliminary communications. The historical and geographical notes were compiled from

this literature. The parts which refers to the parasite and its evolution gives my personal, indepent and generally repeated observations, accompanied by original drawings. Where the work of the commission under LEIPER had already cleared the ground, my personal observations agreed in all the essential points. The part refering exclusively to the local conditions in Brazil is my own. The serial sections used for this paper came from more than fifty blocks and were mostly made in the anatomo-pathological laboratory of this institute, then directed by Dr. OSCAR D'UTRA, and some by Dr. TRAVASSOS of this institute. They were examined by me, as all the others, made in my laboratory without the use of paraffininclusion. The patients who furnished the material for the numerous infections of snails, made by me, were sent by Dr. OSWINO PENNA, after he had observed eggs of *Sch. M.* in their stools.

The *Planorbis olivaceus*, used in my studies, were brought from Aracajú by THEOPHILO MARTINS, sent by the institute, or received from Bahia through the kindness of Prof. PIRAJÁ and Dr. OCTAVIO TORRES. When studying the fresh water shells, I compared the collections in the musea of Rio de Janeiro aud São Paulo.

These studies occupied a great part of my time during more than two years (with many interruptions due to want of the necessary materials). They were helped in many ways by the directors of this institute Drs. OSWALDO CRUZ and CARLOS CHAGAS. To them and the other colleagues already mentioned my best thanks are given.

In 1917 this paper was practically concluded and the plates were ready, but printing was adjourned in order to present the work at the medical congress held in Rio in October 1918. The essential part was read in a lecture, accompanied by projections before an audience, already somewhat reduced by a sudden epidemic of influenza. This and other circumstances further delayed the printing. Since this time only a few additions have been made, in connection with the more recent literature.

2. (Pg. 109, l. 6 from the bottom of col. 1.) *Schistosoma*, derived from σχιστός split and σῶμα body, is the Greck name for the worm and this gives *Schistosomatosis* as correct form.

3. (Pg. 110, l. 11 from top of col. 1.) The blood fluke named by SAMBON *Schistosomum Mansoni* is a good example of the difficulty of nomenclature. The generic name of DIESING is not only more caracteristical but really anterior, though the record of the session in which it was announced, was published after the first paper of WEINLAND. The name of *haematobium* included two species, of which one was first considered new by HARLEY who named it *capense*. It seems to me that this name ought to stand. If the author did not recognize that his speies was part of *haematobium*, he had the excuse that the double shape of the egg in the same specimen of the egyptian blood fluke was considered established by an observation of BILHARZ. This observation, used as principal argument by the unitarian, was most probably erroneous, as may be easily supposed when we take in account that *Mansoni* has only one egg with shell. It has not been confirmed by any recent author and is invalidated by countless observations. If the name of HARLEY holds good, then the name of *haematobium* must be restricted to SAMBON'S *Mansoni* and might mean both species or each one of them. This might be prevented by suppressing the name *haematobium* and distinguishing between *capense* and *Mansoni* but this would be considered incorrect by many and would substitute a little known name for another already much used. In this emergency I shall continue to use the uame *Mansoni*, while the form with an apical spine on its egg wonld be *haematobium* (forma *capense*).

4. (Pg. 111, l. 7, from the top of col. 1.) *Schistosomum* and allied genera in mammalia and birds: There is a considerable number of species which I enumerate in chronological ordre with their synonyms.

1851 *Schistosomum haematobium.* Man and other mammalia.

1854 *Distomum canaliculatum* RUD. (=*Ornithobilhazia* de ODHNER) in spec. of *Larus* and *Sterna.* Found in Brazil by NATTERER, reobserved more than twenty years ago by LUTZ in Santos and since in Rio by FARIA and TRAVASSOS.

1864 *Distomum capense* HARLEY (=*Sch. haematobium* ex parte.)

1876 *Schistosomum crassum* (SONSINO) In cattle and sheep, Africa, Sicily and Sardinia.

1895 *Bilharziella polonica* (KOWALEKY) In ducks. Europe.

1904 *Schistosomum japonicum* KATSURADA. Man and other mammalia.

1907 *Schistosomum Mansoni* SAMBON (*Sch. haematobium* ex parte).

1906 *Schistosomum spindale* MONTGOMERY. Bovidae. India. Sumatra.

1906 *Schistosomum indicum* MONTGOMERY. Bovidae. India. Sumatra.

1906 *Schistosomum bomfordi* MONTGOMERY. Bovidae. India. (France).

1916 *Schistosomum turkestanicum* SKRJABIN. Bovidae. Turkestan.

There are a few more species in birds like *Gigantobilharzia acotyla* ODHNER 1910, *Ornithobilharzia intermedia* ODHNER from European *Laridae* and *Kowaleskii* PARONA from *Larus melanocephalus* in Europe.

These blood flukes form a group of digenetic trematodes to whicch some authors give family value, calling them *Schistosomidae* or *Bilharziidae.* They were discussed under this name by ODHNER (Zool. Anzeiger 1913, p. 58).

5. (Pg. 111, l. 15 from top of col. 1.) V. HOLCOMB 1907. A quite likely explanation is that the female of one species might be carried by the male of the other to its place of predilection thus causing an abnormal localisation of the female.

The terminal prolongation of some eggs of *Sch. m.* (fig. 11) may also be erroneously taken for a spine.

6. (Pg. 112, l. 17 from top of col. 2) I saw this swamp in its natural state which seemed favourable for the breeding of aquatic molluscs. Dr. COSTA LIMA who directed the drainage stated to me that the workmen always accused itching after having entered the water.

7. (Pg. 115, l. 9 from the bottom of col. 2.) After my work was finished I received two interesting papers by WILLIAM CORT (Univ. of Calif. Publ. in Zool. XVIII, N° 17 & 18, Jan. 4, 1919) on the Cercaria of the *Sch. japonicum* and the eggs and miracidia of the other human blood-flukes. In the first paper the author represents the excretory system with its ramifications, the nervous system and a cephalic gland inside of the oral sucker and the rudimentary intestine in very clear (but evidently very schematic) drawings which makes the comparison with *Sch. M.* rather difficult. The second paper contains two microphotographs and five drawings of the eggs and miracidia of *Sch. M.* and *Sch. japooicum.* It is interesting to compare the want of details in the photographs with the distinctness of the drawings (which illustrate the interpretation of the author but hardly resemble the aspect of the microscopical preparations). I call attention to the fact that in his two first cases the author tailed to find the rudimentary spines on the eggs of *Sch. jap.* In a third case they were found in varying size but only in one half of the number examined. The author also gives historical notes and a list of publications.

8. (Pg. 120, l. 13 from the bottom of col. 1.) On the relation between the terminal-spined and lateral-spined eggs of *Bilharzia* Brit. Med. Journ. March 18th 1916, p. 411. "In the form derived from *Bullinus sp.* the males have four or five large testes and the two lateral gut branches are late in uniting, so that even when mature the worms have a short intestinal coecum. In the female the ovary lies in the latter half of the body. The uterus is very long, voluminous and contains many terminal-spined eggs some of which lie

in pairs. The yolk glands have a limited range in the posterior fourth of the body. These worms belong to the species *Schistosoma haematobium* (*sensu stricto*). In the worms derived from *Planorbis Boissyi* the males are small and have eight small round testes. The two lateral gut branches unite very early. In some of the smallest specimens found this union had already taken place. The intestinal coecum is correspondingly very long. The female has the ovary in the anterior half of the body. The uterus is very short, and almost invariably there is only one egg at the time in each specimen when a number have already been laid. The yolk glands are extensive ranging through the posterior two-thirds of the body, along the whole length of the coecum. The eggs always have a lateral spine, the first laid is smaller than those succeeding and the spine is then set almost at right angles to the axis. Pending a consideration of the claims of other names to priority the speciffic name *Schistosomum Mansoni* may be adopted rightly for these worms. They differ in their adult structure from *Schistosoma haematobium* (sensu stricto) more markedly than does *Schistosoma bovis*.

Vesical bilharziosis and Manson's intestinal bilharziosis are therefore etiologically properly regarded as entirely different diseases".

9. (Pg. 123, l. 18 from the bottom of col. 2.) The study of the literature on *Sch. jap.* shows that the disease due to this blood fluke is generally more acute, intense and severe. This is easily understood as the production of eggs is much larger. Also the use of human excrements for fertilizing, the cultivation under water and the prolonged exposure of the workers in flooded ricefields favour intense infection, so that there is no need to suppose a greater virulence of the parasite.

10. The rests of the digested blood must be rejected by the mouth, as in all the trematodes without anus. We also find principally in severe experimental infection of animals black masses as well in the blood, as in the leucocytes and phagocytes in the tissue. They are also seen in human infections where however they might be taken for malarial pigment which in the laboratory animals may be excluded.

11. (Pg. 124, l. 1 from top of col. 1.) Lately (1918) LAWTON described an epidemic of fevers, observed in Australian soldiers, infected in Egypt. The symptomatology was very much alike with that of intense and acute infections by *Sch. jap.*

12. (Pg. 125, l. 1 from the bottom of col. 1.) We have in the institute a film showing a white rat, after prolonged immersion of the posterior part of the body in water with many live cercariae. The itching principally in the hind feet and tail is charactically shown by the biting of these parts.

13. (Pg. 124, l. 19 from the top of col. 2.) I record here that in the acute infections, cited in note 11, there was cough, accompanied by evident symptoms of bronchitis and pulmonary congestion. The sputa sometimes contained blood. These are early symptoms of infection. Later ones might be due more to the eggs than to the worms.

14. Microscopical preparations of spontaneous infection with *Sch. Jap.* in the large domestic animals show distinctly the latter lesions, cirrhosis of the liver and alterations in the veins.

15. Considering that anthelmintyics may not reach the flukes in the portal circulation, unless extremely diluted, other remedies were tried, such as arsenical and antimonial compounds, emetine and methylene blue. However, these substances although useful in infection, by protozoa, spirilla and allied organisms, dont seem to promise much result when used against blood-worms. Even if they killed the flukes without producing new and perhaps even more serious symptoms, there would still remain the eggs and the lesions caused by them, unless the infection was quite recent and therefore not easily recognized. Several authors claim good results, but such ought to be demonstrated by animal experiments which are easily made. LEIPER who

proceeded so, failed to get good results. The literature of this question may be found in a thesis of ELPIDIO DE ALMEIDA, just published (1919).

Additional Note (Jan. 1920): CHRISTOPHERSON published several papers on the treatment of *Schistosomum* infection by Tartrate of Antimony. One of them, in collaboration with J. R. NEWLOWE, appeared in the Journ. of Trop. Med. V. XXII, pg. 128, 1919 and refers seventy treated cases. He declares that the results were very good and were partly controlled for two years. The author mentions mostly cases of *Sch. haematob.* and recommends a dose of 25—30 grains, approximately 2 grammes. The first injection contains ½ grain in 3 ccm. of physiological salt solution. (The injections are made in a vein.) This is repeated every second day, always increasing the dose by half a grain, till 5 grains are reached. After some days the blood disappears from the urine and the eggs become sterile about two weeks after the dose of 20 grains has been reached.

These results want more confirmations refering to patients with *Sch. M.* One experience we made in a guinea pig shows that a cure cannot be obtained by a few large doses. The treatment must be necessarily rather long, when made by this method, and may not be shortened, as the drug must be used with precaution.

Explanation of Plates.

Plate 37 (Figs. 1—8).

Fig. 1 ♂ & ♀ of *Sch. haematobium*. The ♀ contains many eggs with terminal spine.×28. Copied from FRITSCH.

« 2 ditto. The ♀ shows the intestine double till near to the caudal end. Copied from BILHARZ.

« 3 ♂ & ♀ of *Sch. Mansoni*. The balsam-preparation shows clearly the bifurcarted intestine which in both sexes unites before the middle of the body.×50. Orig.

« 4 ♀ of *Sch. M.*, first third of the body, showing only one egg with lateral spine.×40. Haematoxylin stain. Orig.

« 5 ♂ of *Sch. M.* showing the testicular apparatus.×20. Orig.

« 6 Another ♂ of *Sch. M.* showing the same in three different positions.×60. Orig.

« 7 a, b. Two ♂♂ of *Sch. M.* showing the common appearance of eight testicular lobes. No sign of a vesicula seminalis.×60. Orig.

« 8 of *Sch. haematobium* copied from BILHARZ.

Plate 38 (Figs. 9—38).

Fig. 9—14 Abnormal eggs of *Sch. M.* 9—11 Shell with protruded anterior pole, liable to be mistaken for a terminal spine; 12 egg with two spines; 13 crystals deposited on egg shell; 14 calcified egg. ×150. All the eggs were voided by patients infected in the north of Brazil.

« 15 Normal egg of *Sch. M.* showing the embryo.×400.

« 16 Miracidium observed some time after ecdysis.—Stained preparation.×400.

« 17 Miracidium which failed to penetrate a long time after ecdysis.×400.

« 18 Miracidium attacking the antennae two have already penetrated.× 200.

« 19 *Planorbis olivaceus* with normal, 20 & 21 with infected antennae. The specimens are halfgrown. Natural size.

« 22—38 Aspect of antennae in various degrees of infection. Figs. 19—27 in nat. size, 29-38 somewhat enlarged. Fig. 32 shows a normal antenna. All the drawings are original and, with exception of 32, taken from fresh or living specimens.

Plate 39 (Fi s. 39—47).

Fig. 39 a young sporocyst inside of the antenna showing the 4 tubular orifices with vibrating membrane and the ciliated epithelium half detached, b the same more developed with initial state of secundary sporocysts.×100.

« 40 Almost mature primary sporocyst, the young second generation escaping through a tear of the antenna under compression.×150.

« 41 Section through an antenna containing an aimost mature primary sporocyst. Haematoxylin staining ×150.

« 42 Secondary sporocyst.×500.

« 43—45 Secondary sporocysts, more developed, from the internal organs of Planorbis. Lateral illumination.

« 46—47 Ditto, seen by transparency. All the drawings are original and taken from fresh preparations, with exception of 41.

Plate 40 (Figs. 48—63).

« 48 Section of liver of an infected Planorbis showing an almost ripe cercaria in longitudinal section- Stained preparation.×250.

« 49 & 50 Ditto showing cuts of secondary sporocysts.×700.

« 51 Living cercariae of *Sch. M.*×30.

« 52—61 Cercariae of Sch. m. from various preparations.×120.
« 62 Living cercaria. The drawing is combined from various.
« 63 Stained section showing the twisted excretory ducts and the superior gland cells in oblique cut.

Plate 41 (Figs. 64—70).

Fig. 64 *Dicranocercaria ocellifera* alive×60
« 65 Ditto stained (glycerinpreparation).×250.
« 66 Ditto unstained×250.
« 67—68 *Dicranocercaria valdefissa.* Unstained preparation×250.
« 69 Section of skin from pig showing a penetrated *Schistosomulum.* Stained preparation.×600.
« 70 Intestinal loop of infected guinea pig showing mesenteric veins dilated and full of flukes. Formolfixation. Natural size.

Plate 42 (Figs. 71—73).

« 1, 2 Sections of livers of infected snails.
« 3 4 Sections of mesentery of infected guinea pig showing the flukes in the veins in longitudinal and transversal cut.
« 75 Liversection from guinea pig including a transversal cut of female *Sch. M.*

The figures show photographs taken from stained preparations by J. PINTO, photographer of the institute.

Plate 43 (Figs. 1—4).

Fig. 1 a—d Adult specimen of *Planorbis olivaceus.* Shell in dorsal and ventral aspect, in profil and in transverse section; f adult specimen with upper half of shell removed, exposing the animal (combined drawing), e section of a small and aberrant specimen. Nat. size.
« 2 Shell of adult *Planorbis guadeloupensis*, a dorsal, d ventral aspect, b transversal section, c natural size.
« 3 The same refering to *Pl. centimetralis.*
« 4 The same refering to *Planorbis Boissyi.*

In the plate the sections in horizontal position show the mouth to the right. If we consider the shell sinistral, the ventral side was turned upwards and the dorsal downwards. The section in vertical position show the mouth below.